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RESTORATION OF BLOOD FLOW IN DAMAGED ARTERIES

FURTHER STUDIES ON A NONSUTURE METHOD OF BLOOD VESSEL ANASTOMOSIS

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THERE WERE 4,403 U. S. soldiers who lost one or more extremities[1] during the last World War, and of this number only 13 per cent (690)[2] lost their limbs in battle. Of the remaining 3,713 cases, surgical amputation was resorted to. The majority of these surgical amputations were performed because of infection or because of the presence of serious damage to the blood supply, or both. Attempts at suture of severed arteries was "by-and-large" unsuccessful and, therefore, considered impractical.[3, 4, 5]

Perhaps reflecting the improved methods of treating wounded extremities in the present war, a recent British war memorandum by Admiral G. Gordon-Taylor, and his associates,[6] has the following to say regarding amputation: "The main indication for amputation is irreparable interference with blood supply. No matter how severe the destruction of skin, comminution of bones or contamination of tissues, if the main blood vessels are not destroyed, the limb can usually be saved."

We postulate in this war that the use of the sulfonamides in conjunction with careful débridement, careful hemostasis with the evacuation of blood clot will, for the first time in the history of wars, enable the surgeon to safely postpone amputation in cases of ligated primary arteries until the true status of the collateral circulation is established. Furthermore, such measures, perhaps in conjunction with the use of anticoagulants, will afford the basis for success of a simple, nonsuture method of arterial anastomosis.

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We should like to report our experience using the vitallium* tube, or tubes, as a prosthesis for blood vessel anastomosis.

METHODS

Several different technics (Figs. 1, A and B, and 2 A) were attempted before a satisfactory method was discovered. In brief, the suture of the divided carotid (or femoral) artery over a vitallium tube was successful

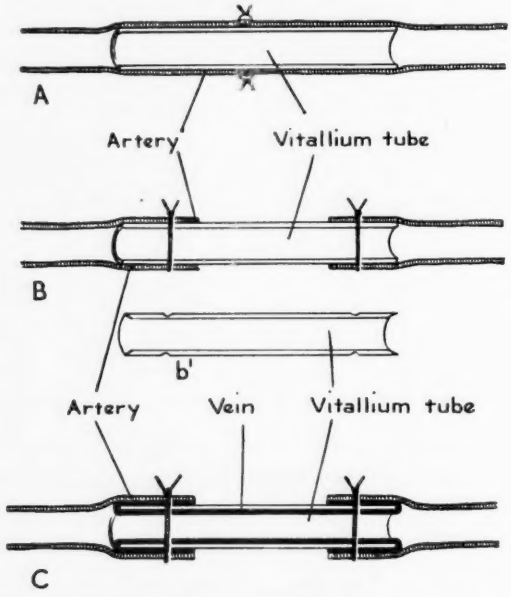

FIG. 1.—In these drawings are depicted the different methods of using a single vitallium tube. (A) The artery is sutured in the usual manner over a straight vitallium tube. (B) The divided ends of the artery are approximated near each end of the vitallium tube by ligatures, in this way a gap is bridged. (C) The final technic which was highly successful. The drawing shows the vein to be inside the vitallium tube; the ends of the vein are everted over the vitallium tube, and the artery is drawn over each end and held by a ligature. In this way the flowing blood contacts only an intimal-lined tube.

in only one out of nine experiments, and in the successful one blood flow was demonstrated for only five days (Fig. 1 A). Bridging a gap with a vitallium tube by the ligature technic (Fig. 1 B) was also most disappointing (none out of six). Finally, the bringing of each end of the divided artery through a vitallium tube and bridging the gap with a vein transplant was successful in only two cases out of seven (Fig. 2 A).

After the above failures, a technic was established of bridging a gap in an artery which functioned fairly well in the small femoral artery and with

* The approximate composition of vitallium is cobalt 65%, chromium 30%, and molybdenum 5%. The tubes were supplied by the Austenal Laboratories, Inc., of Chicago and New York. We wish to thank Mr. J. J. Erdmann of that company for his cooperation.

uniform success in the abdominal aorta for an indefinite length of time (Figs. 1 C and 2 C). Dogs have been used as the experimental animals, and silk technic was used throughout. No anticoagulant was employed. Vitallium tubes 2.5 cm. long, with an outer diameter of 3 Mm. and an inner diameter of 2 Mm., have been used as a permanent prosthesis for a vein transplant. The operation (Fig. 3) is carried out as follows: The femoral artery and vein of the dog are located and isolated for distances of 7 to

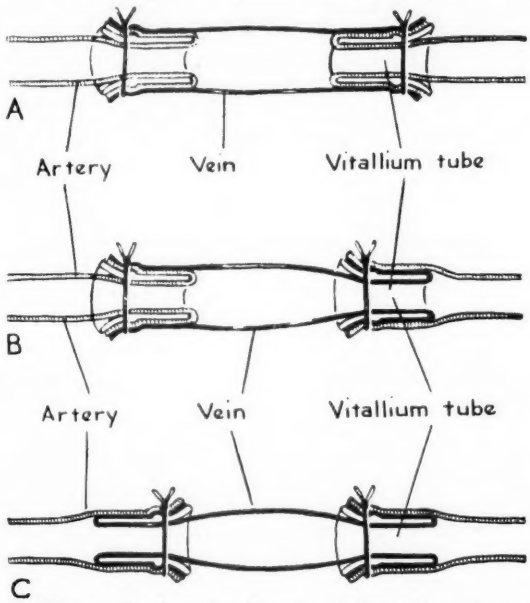

FIG. 2.—In these drawings are depicted the different methods of using two vitallium tubes in combination. (A) The divided ends of the artery are brought through each vitallium tube and everted; a ligature holds the everted artery in place; a vein transplant is then brought over each vitallium tube and held with ligatures. (C) The vein is brought through each vitallium tube and the everted ends held in place by ligatures. The ends of the divided artery are brought over the vitallium tube and held in place by ligatures; this method makes it possible to bridge a gap of any length, the limiting factor being the length of the vein transplant. (B) A combination of the methods shown in (A) and (C).

8 cm., each branch being ligated close to the vein with arterial silk and loosely to the artery with fine silk. The segment of vein, 7 cm. long, is then excised between suture ligatures and promptly irrigated with saline solution through a blunt-nosed eye dropper. The distal end of the vein is identified so that it will be brought into approximation with the proximal end of the femoral artery, allowing blood to flow through the graft in the direction of the valves. One end of the vein is then pushed through the inside of the vitallium tube and the ends of the vein are everted over the tube for 1 cm. at each end. A single tie of fine silk near each end holds the vein in place. The tie is placed some 8 to 9 Mm. from the end of the tube. Care is taken from this point to allow nothing to touch the exposed intima of the vein and it is frequently moistened with saline solution. The femoral artery is then

divided after rubber-shod clamps have been applied. The ends of the artery are irrigated with saline solution and, with three mosquito clamps, each end of the artery is brought over the vitallium tube lined with vein for the distance of 1 cm. The ends of the artery are fastened to the tube beyond the ridges with two ties of fine silk at each end (Fig. 1 C). This maneuver

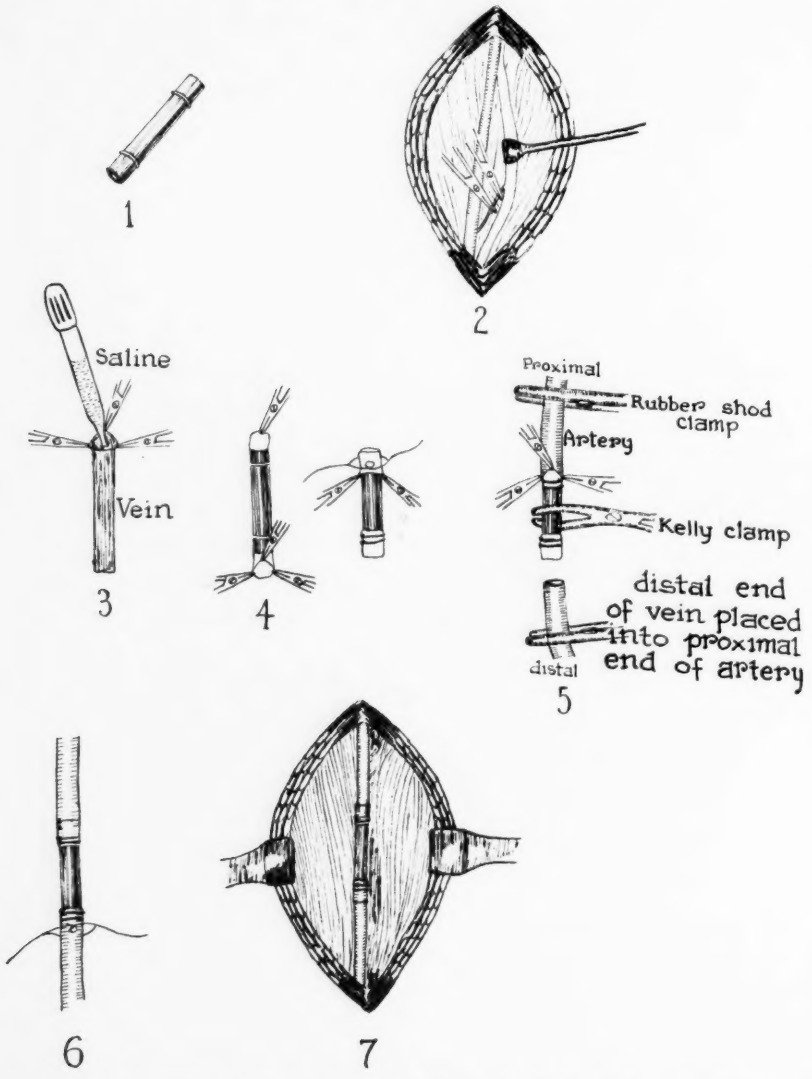

FIG. 3.—The various steps in the operation as carried out in the femoral artery. (1) The vitallium tube with its two ridges (sometimes grooves). (2) Shows the artery and vein exposed; the vein is retracted and clamps have been placed upon a branch. We now think it is perhaps better technic to ligate the branch first, clamp distally, and cut between. (3) The segment of vein upon removal is irrigated with saline solution through a blunt-nosed eye dropper. (4) The vein has been pushed through the inside of the vitallium tube; the two ends are everted over the ends of the tube and are held in place with one or two ligatures of fine silk. (5) The distal end of the segment of vein is placed into the proximal end of the artery and held there by two ligatures of fine silk; all ligatures are placed behind the ridges. (6) The snug ligature near the end of the vitallium tube, for the apposition of the artery and vein, is being tied. (7) The completed operation showing the femoral artery—a gap of two centimeters has been bridged.

results in the intima of the artery being in contact with the intima of the vein for 6 to 8 Mm. Finally, a tie of fine silk is tied loosely over the artery at each end, 1 to 2 Mm. from the end of the vitallium tube, so that blood cannot penetrate between the two intimas (Fig. 4) and, hence, form a thrombus in the first place; and, secondly, so that tissue juices released by the very tight ligatures cannot escape into the circulation. After the above has been completed the proximal rubber-shod clamp is released first in order that air bubbles will be driven peripherally. Immediately after release of the distal rubber-shod clamp the blood courses freely and a strong pulsation can be felt distal to the vitallium tube.

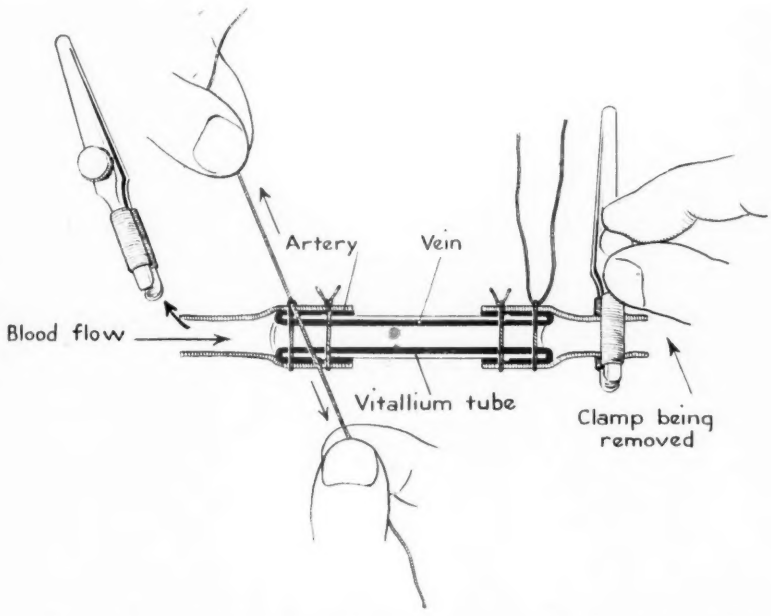

FIG. 4.—Illustrating the tying of the snug ligature near the end of the vitallium tube. This ligature prevents blood from penetrating between the two intimas and also keeps any tissue juice, freed by the crushing ligature, from escaping into the general circulation.

A modification of the above procedure has been made for the use of longer gaps in an artery. Two vitallium tubes with funnel ends are handled in a similar fashion to the above method (Fig. 2 C).*

We have used the nonsuture method in the anastomosis of the dog's abdominal aorta with regular success, grafts from the external jugular vein being employed to bridge the defect. Several of these animals have been followed now in excess of seven months, and their anastomoses have continued to function perfectly.

Clinically, we have used the two-tube method to bridge a combined defect of the popliteal and femoral arteries in a 63-year-old negro following

* For a more detailed description of the method refer to Surgery, **12**, No. 3, 488–508, September, 1942.

excision of a syphilitic aneurysm. In spite of the presence of marked degenerative arteriosclerosis, gangrene of the leg was avoided.

Irrespective of the above encouraging results, it is our opinion that the acid test of the method is the study of its efficiency when used in anastomosing the small femoral arteries of dogs in clean and contaminated wounds, and without the use of anticoagulants.

The femoral artery of the dog was selected as a vessel for testing the efficiency of our nonsuture method of anastomosing vessels for the following reasons: (1) Abundance of experience has shown that suture anastomoses of the femoral arteries of dogs, with or without the use of vein grafts, fails

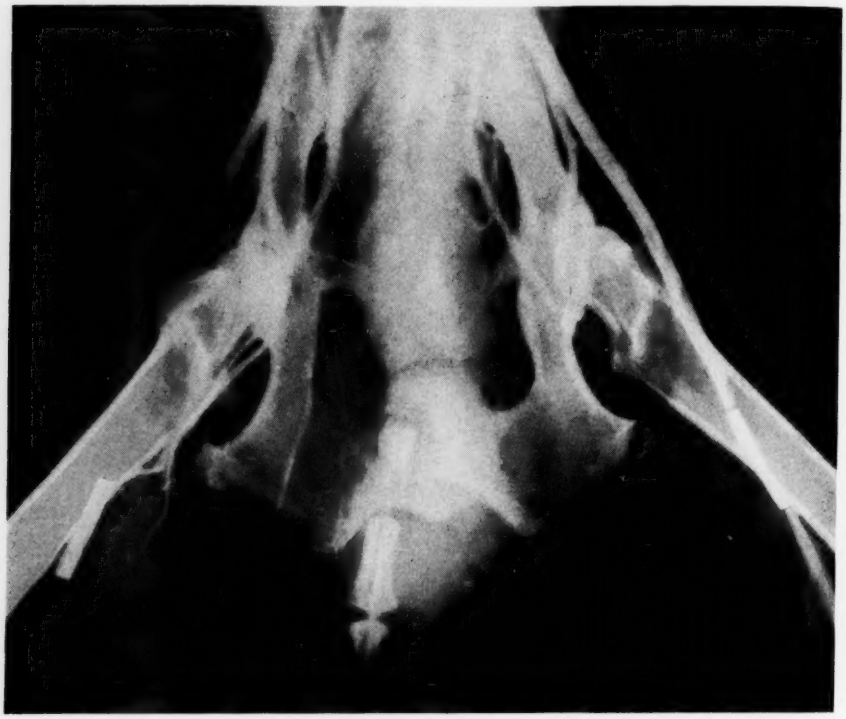

FIG. 5.—(A) Diodrast injection of the abdominal aorta in animal No. 1862 shows the right femoral artery to be entirely patent; the left side, however, is blocked some eight to nine centimeters above. This roentgenogram was taken 69 days postoperatively.

as often as it succeeds even when undertaken by skilled operators under rigid aseptic precautions, whereas the same technic may be employed to anastomose the aorta or carotid arteries with fairly regular success. (2) A review of the literature on the subject of methods employing a nonsuture prosthesis for the anastomosis of blood vessels reveals not a single instance of success in bridging defects of the femoral artery in dogs using vein grafts.

In Figure 5 is shown an arteriogram of a dog's femoral vessels 69 days after anastomosis of each femoral artery using the single tube method. The anastomosis of the right femoral artery is evidently patent, whereas thrombosis has taken place on the left, although this anastomosis had been proven

to be patent on exploration 33 days after anastomosis had been performed. Figure 6 is a photograph illustrating a probe through the excised anastomosis of the right femoral artery. The sheath of the femoral artery has healed about the tube without evidence of irritation by the vitallium, and the tube can be seen shining through the thin sheath of the artery.

Figure 7 is a photograph of a specimen of an anastomosis of the femoral artery excised 35 days after the insertion of a vein graft over a single vitallium tube. The latter has been removed and one end of the anastomosis opened to expose the smooth healing of the intimas of the artery and vein. A photomicrograph of the previous specimen shows that the intima has bridged the artery-vein junction (Fig. 8). Firm healing has occurred between the artery and vein. The vein graft appears essentially normal and well nourished,

FIG. 6.—Specimen of the right femoral artery of animal No. 1862 showing minimal reaction around the vitallium tube, and the silk ties to have remained in place; complete healing has occurred between the intima of the vein and that of the artery at each end.

and this is important because of the fact that in the single tube method of anastomosis the vein graft is completely isolated from the perivascular tissues.

The use of a vein is the only practical means of successfully bridging a sizable arterial defect. Therefore, a method of anastomosing arteries to be of service in the war-wounded, and of maximum service clinically, must, of necessity, be easily adaptable to the use of vein grafts. Such is not the case with the suture method under war conditions. Increasing experience with nonsuture method, with strict attention to details of technic and asepsis, and the use of generous segments of femoral vein from the opposite leg, affords a 90 per cent expectancy of success in bridging defects in the small femoral arteries of dogs.

ANASTOMOSIS OF BLOOD VESSELS IN CONTAMINATED WOUNDS

Experience has shown that infection is the greatest single cause of failure in any method of blood vessel anastomosis. It has long been known that the wound may heal by primary union and yet cultures taken from the site of the anastomosis may reveal bacterial growth. Experiments were conducted in which the Carrel suture technic was employed to anastomose free vein transplants to the divided ends of the femoral arteries of dogs (Table I). The operation was performed six hours after unsterile ligation and division

TABLE I

RESULTS WITH CARREL SUTURE TECHNIC EMPLOYING FREE VEIN TRANSPLANT BETWEEN DIVIDED ENDS OF FEMORAL ARTERY
OPERATION PERFORMED 6 HOURS AFTER UNSTERILE LIGATION AND DIVISION OF FEMORAL ARTERY

Perfect Function and Structure (14 Days)	Perfect Function but Aneurysm (14 Days)	Thrombosed (14 Days)	Secondary Hemorrhage (3–7 Days)
1	3	2	4

of the femoral artery. One and one-half grams of sulfanilamide was used in alternate wounds in the series, and all of the wounds were thoroughly irrigated before performing the anastomoses. Of the ten anastomoses, one was found to be successful upon exploration 14 days postoperative, while aneurysm occurred at the site of the anastomosis in three instances, thrombosis occurred in two, and secondary hemorrhage in the remaining four.* Figure 9 is a photograph of the specimens showing aneurysms at the site of the anastomoses. Cultures taken from the aneurysms revealed bacterial growth, as was the case in the rest of the failures.

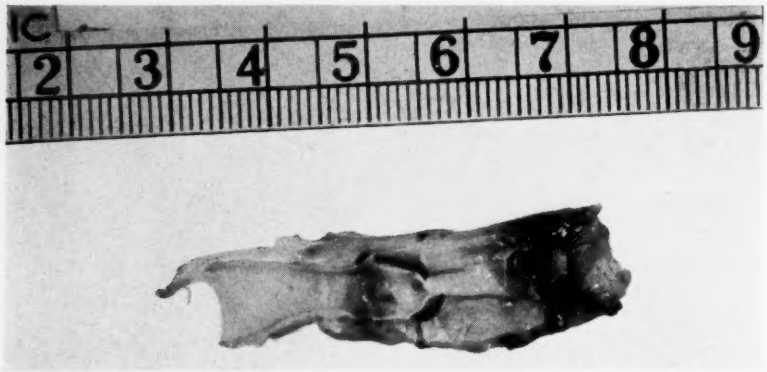

FIG. 7.—A photograph showing the opened specimen of the left femoral artery, where excellent healing has taken place between the intimas of the artery and vein. The vitallium tube has been removed and the vessel is entirely patent; this having been demonstrated by sectioning the artery distal to the anastomosis. It is interesting, however, that a diodrast injection made just before the animal was sacrificed failed to demonstrate patency.

Table II shows the results with the nonsuture method of bridging arterial defects in the femoral arteries of dogs using the double tube vein graft technic. The operation was performed six hours after unsterile ligation and

TABLE II

RESULTS WITH NONSUTURE METHOD OF BRIDGING ARTERIAL DEFECT IN FEMORAL ARTERY
OPERATION PERFORMED 6 HOURS AFTER UNSTERILE LIGATION AND DIVISION OF FEMORAL ARTERY

Perfect Function and Structure (7 Days)	Perfect Function but Aneurysm (7 Days)	Thrombosed (7 Days)	Secondary Hemorrhage (7 Days)
12	0	10	0

division of the artery, and, again, 1.5 Gm. of sulfanilamide was used in the wounds of alternate dogs. Of the 22 anastomoses performed, 12 were successful, and thrombosis occurred in 10.*

* The anastomoses were explored on the 7th postoperative day because experience has shown that if the anastomoses remain patent up to the 7th day, thrombosis is extremely unlikely to occur thereafter, whereas with suture anastomoses failure is likely to occur up to 14 days after operation.

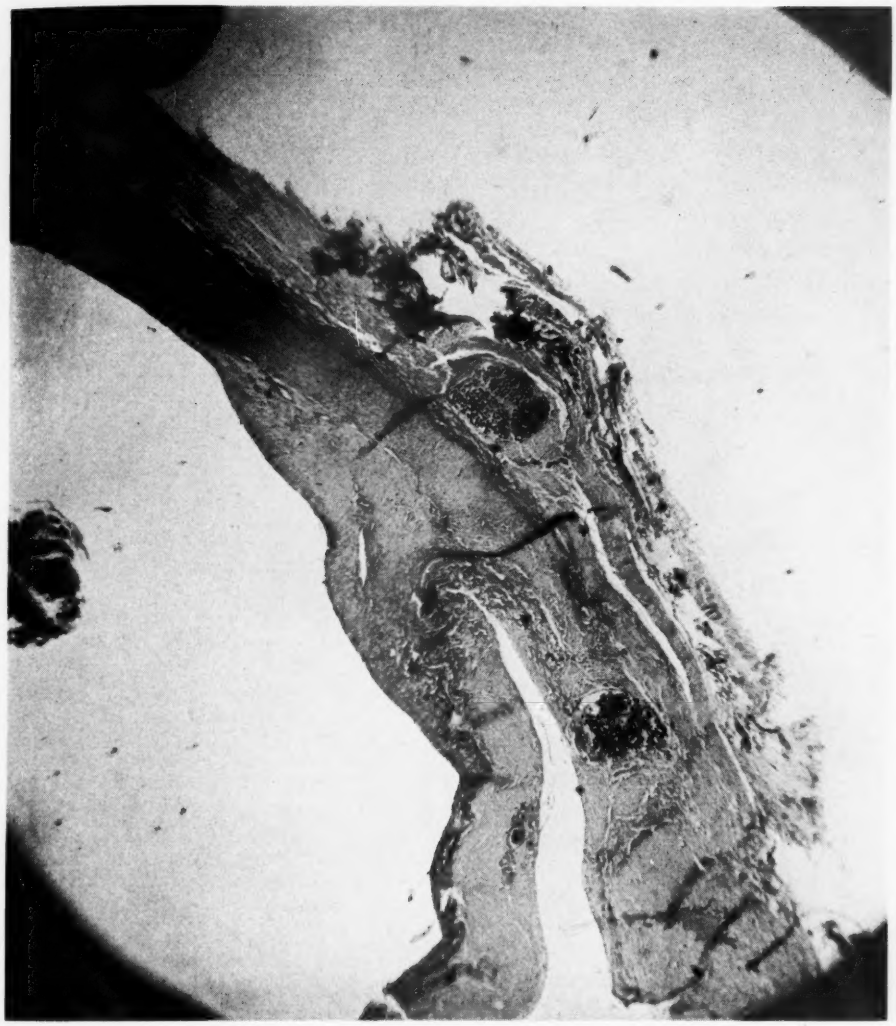

FIG. 8.—Photomicrograph of specimen removed from animal No. 1880 showing the junction of the artery and vein. This junction has been bridged by intima.

A comparison of these two series of animals reveals the dire results of suture anastomoses of arteries in the presence of bacterial contamination. It is of interest to note that sulfanilamide was used in the one successful experiment, whereas with the nonsuture method, using vitallium tubes, the anastomoses succeeded in over 50 per cent of the experiments in spite of bacterial contamination. In the failures, unlike the suture method, simple thrombosis occurred without secondary hemorrhage.

Table III: In this series of ten animals the following changes were made:

1. The performance of the anastomosis was delayed 24 hours after unsterile ligation and division of the femoral artery.

2. The dirty wounds were débrided before performing the nonsuture anastomoses.

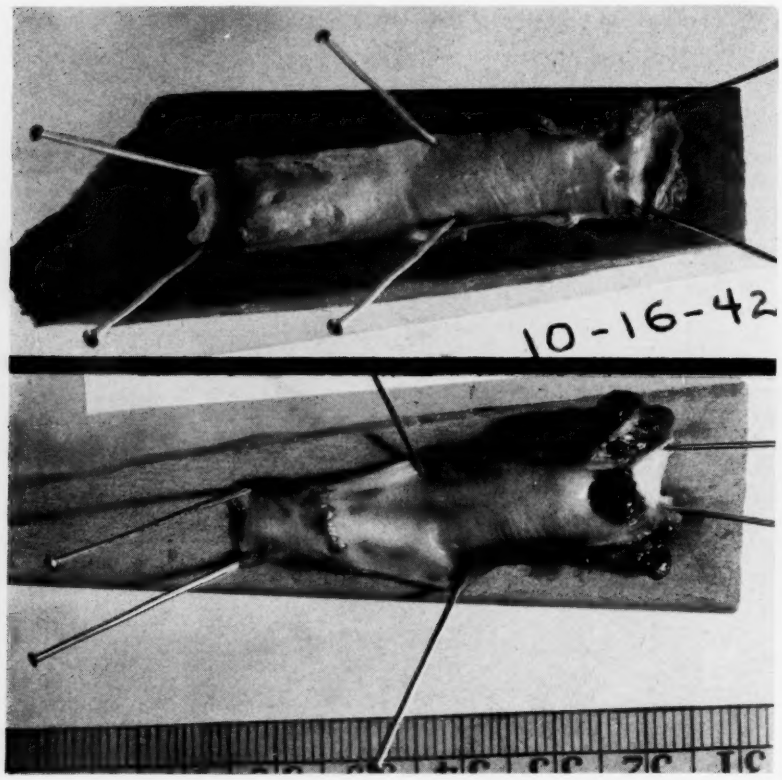

FIG. 9.—Specimens showing aneurysms at the sites of anastomoses; note the aneurysms in the upper right of animal No. 1987 and the lower right of animal No. 2018. This latter one is almost completely filled with thrombus.

3. Sulfanilamide was placed in the wounds of alternate dogs.

4. The vein graft was taken from the opposite leg, and in generous lengths, to avoid tension upon the anastomosis.

Results: Success in three of ten anastomoses.

TABLE III

RESULTS WITH NONSUTURE METHOD OF BRIDGING ARTERIAL DEFECT IN FEMORAL ARTERY
OPERATION PERFORMED 24 HOURS AFTER UNSTERILE LIGATION AND DIVISION OF FEMORAL ARTERY
NO SULFATHIAZOLE

Perfect Function and Structure (7 Days)	Perfect Function but Aneurysm or Partial Thrombosis (7 Days)	Thrombosed (7 Days)	Secondary Hemorrhage (6 Days)
3	1	5	1

The successes in this group of 24-hour delayed anastomoses three out of ten represents a reduction of 25 per cent when compared with 12 out of 22 successes in the six-hour delayed anastomosis group of Table II. This "fall off" in success occurred in spite of the fact that in the 24-hour group the wounds were carefully débrided immediately before performing the anastomoses.

Whereas the gross appearance of the wounds of the two series differed little during healing, the behavior of the anastomoses in the two groups

reflected, with remarkable sensitiveness, the true bacteriologic status of the two groups of wounds, namely, the state of bacterial contamination in the six-hour group and spreading infection in the 24-hour group. It is fair to state, however, that sulfanilamide was placed in the wounds at the time of the performance of the anastomoses in two out of the three successes in the 24-hour group.

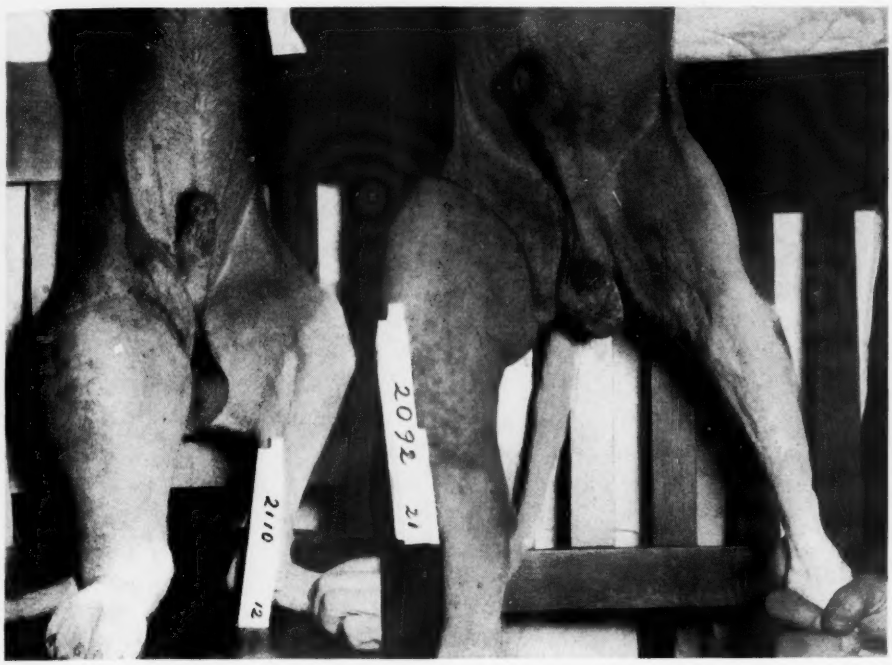

FIG. 10.—Photograph of two dogs, No. 2110 (12 days) and No. 2092 (21 days) postoperatively; in which the right hind leg was amputated at the midthigh level and reimplanted after having been kept in cracked ice for 24 hours. Moderate edema is still present in the 12-day animal.

Table IV: The experiments in this series of ten dogs are identical with the former series (Table III) except that each dog received one gram of sulfathiazole by mouth twice daily from the time of the initial unsterile wound until the day of examination of the anastomosis one week later. Results: Success in eight out of the ten anastomoses, and one of the two failures was thought to be due to a technical difficulty in dealing with a venous valve.

TABLE IV

RESULTS WITH NONSUTURE METHOD OF BRIDGING ARTERIAL DEFECT IN FEMORAL ARTERY
OPERATION PERFORMED 24 HOURS AFTER UNSTERILE LIGATION AND DIVISION OF FEMORAL ARTERY

Perfect Function and Structure (7 Days)	Perfect Function but Aneurysm (7 Days)	Thrombosed (7 Days)	Secondary Hemorrhage (7 Days)
8	0	2*	0

Table V is identical with the former series (Table IV) except that Carrel suture anastomoses were performed in the ten animals. Results:

* One of these two was transporting blood satisfactorily but after removal examination revealed partial thrombosis to have occurred within the vein near the proximal tube.

Four out of ten anastomoses were completely successful. Thrombi were found at the site of the anastomoses in four cases, complete thrombosis in one, and secondary hemorrhage in one case.

TABLE V

RESULTS WITH CARREL SUTURE TECHNIC EMPLOYING FREE VEIN TRANSPLANT BETWEEN DIVIDED ENDS OF FEMORAL ARTERY
OPERATION PERFORMED 24 HOURS AFTER UNSTERILE LIGATION AND DIVISION OF FEMORAL ARTERY

Perfect Function and Structure (14 Days)	Perfect Function but Aneurysm or Partial Thrombosis (14 Days)	Thrombosed (14 Days)	Secondary Hemorrhage (4 Days)
4	4	1	1

In addition to the simplicity and rapidity of the performance of the nonsuture technic in comparison with that with the Carrel suture, the former succeeds far more regularly in the presence of bacterial contamination. The high percentage of favorable results (80 per cent) obtained with our nonsuture method in anastomosing the small femoral arteries of dogs in 24-hour-old, dirty wounds, encourages us to believe that the method will succeed, when used in conjunction with the sulfonamides, if employed for the anastomosis of severed arteries in the war-wounded. It is our experience, and but logical to conclude, that the use of the method upon larger vessels will contribute to its greater success. It is reasonable to believe that in the clinical application of the method the factors of rest, wound splinting and postoperative care; the elimination of pain and vasospasm; and the possible use of anticoagulants would insure its success.

BASIC REQUIREMENTS OF A METHOD OF ANASTOMOSING BLOOD VESSELS IN THE WAR-WOUNDED

I. The method must afford a high incidence of success when used in badly contaminated wounds up to 24, or more, hours after injury.

II. The method must be adaptable to the use of vein grafts for bridging arterial defects without complicating the technic or efficiency of the method.

III. The method must be simple.

Our experiments with the nonsuture method, when used in conjunction with the sulfonamides, affords convincing evidence that the method will fulfill requirement I. The method, employing the double tube technic, is peculiarly adaptable to the use of vein grafts, thereby fulfilling requirement II, whereas, the use of variable-sized vein grafts complicates the technic and greatly extends the performance time of suture anastomoses. As to requirement III, the very simplicity of the nonsuture method commends its use in surroundings as crude and bare of equipment as the necessity of war may dictate.

It is our sincere opinion that the nonsuture method of anastomosing vessels, as presented, meets all the requirements for successful anastomosis of the severed primary arteries in the war-wounded. The one factor affecting the benefits from the method in this war would be the extent to which it is used. Throughout the history of wars there is no greater dilemma than that

faced by the surgeon in regard to the rationing of treatment to the wounded under the pressure of battle. In the last world war, for example, to what purpose would be the contemplation of a method of anastomosing a severed artery when, on a given occasion, there may be several soldiers whose lives needed saving.

In consideration of a method of saving limbs in this war first thought must then be given to the question to what extent will surgical care be rationed? At the onset of the present European war, because of rapidly shifting battle fronts due to mechanized warfare, it became obvious that the seriously wounded would have to be operated upon and cared for in base hospitals far removed from the front, and the ambulance plane became the solution to the problem of evacuation of the seriously wounded. Once the plane is in the air it does not necessarily have to land its wounded at any one overworked base hospital. The wounded may be flown to hospitals where, under favorable conditions, experienced surgical teams can operate, and where postoperative care can be carried out continuously by the same team. The constantly shifting battle line requires the use of numerous mobile first aid and evacuation units where hemorrhage may be controlled, shock treated, bone splinted, and chemotherapy administered before evacuation of the seriously wounded. The above system has already successfully met the challenge in practice upon the Russian and other battle fronts in this war. It has proven sufficiently swift for the successful treatment of serious cranial, chest and gunshot wounds of the abdomen. It is our opinion that the clinical course of the average case of severe extremity wound with severed primary artery lends itself to successful handling in this manner. The following is a case in point:

Case Report.—A 40-year-old junk dealer was admitted to Presbyterian Hospital in deep shock. Three hours before, the patient had fallen beneath the wheels of his loaded wagon and the wheels had passed over the back of his right leg, causing a crushing laceration of all tissues superficial to the bone just above the knee joint. Following a transfusion of 1000 cc. his blood pressure rose and the wound began to bleed again. The leg which was cold and pulseless began to warm slightly and assume a slightly pink color. Exploration of the hematoma, which had dissected most of the posterior muscle planes of the thigh to below the knee, revealed the retracted ends of the popliteal artery which had been crushed against the femur. Blood clot was thoroughly evacuated. The ends of the artery were ligated with silk. The popliteal vein had been crushed and had become thrombosed. It was ligated above the thrombus with silk. The wound was thoroughly débrided, irrigated with saline, and sulfanilamide was implanted into the wound, which was left open and the leg splinted. Sulfadiazine was given intravenously and continued by mouth.

Subsequent to operation the leg was placed at heart level. The location of the wound precluded the use of the Pavaex boot. The blood pressure was maintained and anemia corrected postoperatively by transfusions, and an alcohol block of the lumbar sympathetic nerves on the right was carried out. The man's pain was controlled by opiates. Under continued sulfonamide therapy, the clinical course of this man from this point on was of extreme interest. In the first place, throughout his entire postoperative course the patient developed no evidence of infection either in the wound or systemically. For the

first 36 hours there was no disagreement on the question of sufficient circulation to the foot to maintain life. The veins emptied freely on slight elevation and filled slowly at heart level. The foot was fairly warm and of pale but living color. After an interval of 48 hours traumatic edema began to register its first deleterious effect upon the circulation, as evidenced by slight obstruction to venous return. These effects became increasingly manifest as venous engorgement, cyanosis and slight edema of the foot and ankle through the 3rd and 4th days after injury developed. But it was not until the 4th and 5th days that a declining temperature of the foot heralded the beginning of strangulation of the arterial collateral flow. From then on the tissue damage from anoxia became rapidly irreversible and gangrene was full blown by the 7th day after injury.

We have presented the above case in some detail because it illustrates well some important clinical facts. In the first place the case is not dissimilar to a war wound. In illustrating the benefits of sulfonamide therapy, the conclusion seems inescapable that the onset of gangrene in this case was greatly postponed by the control of infection. One, likewise, gathers the impression that had there been a little more collateral circulation present, the period of traumatic edema might have been successfully tided over. It was equally obvious that a successful anastomosis of the popliteal artery performed at any time up to the 5th day after this man's injury would have saved his leg. Even after the onset of edema, reestablishment of blood flow through the artery would have permitted elevation of the leg with subsidence of the edema.

It is our opinion that a war wound case of the above type may be successfully handled as follows:

Control of hemorrhage (preferably ligation of the bleeding vessels).

Treatment for shock and administration of chemotherapy by the first aid and evacuation units.

Transfer by ambulance plane to a base hospital for débridement and evaluation of the status of the collateral circulation.

Anastomosis of the primary artery may be undertaken when and if indicated.

Our experience with the nonsuture vein graft method, when used in conjunction with the sulfonamides in anastomosing arteries in infected wounds 24 hours after unsterile ligation, indicates a likelihood of functional success.

HETEROPLASTIC VEIN GRAFTS

By heteroplastic we mean a vein graft transplanted from one subject to another. In contemplating the use of the nonsuture method of anastomosing vessels under circumstances more adverse than the facilities afforded by a base hospital, we concede advantages attending the use of heteroplastic grafts. For example, the time necessary for completion of an anastomosis by an average operator with a vein graft at hand does not exceed 15 minutes. Important questions arise in considering the use of heteroplastic vein grafts for bridging arterial defects in the war-wounded. (1) How long would it

be necessary for an anastomosis to function to prevent the occurrence of gangrene? There is clinical and experimental evidence to support the opinion that if an anastomosis functions beyond the period of posttraumatic edema, up to 14 days, the limb will be saved. Reichert[7] amputated (cutting all tissues except bone, large nerves, femoral artery and vein) and replanted the hind legs of dogs and showed that arteries developed across the replant line on the 3rd postoperative day. Veins and lymphatics regenerated on the 4th and 5th days. He, likewise, proved that the regenerated vessels were functionally adequate on the 8th postoperative day by ligation of the femoral artery and vein without the occurrence of gangrene. However, if the artery itself is ligated alone gangrene of the leg can not be prevented until after the 14th day.

(2) Will heteroplastic vein grafts function adequately when used to bridge vessel defects? To gain information on this important question we performed the following experiments. Figure 10 is a photograph of two dogs in which the right hind leg was amputated at the midthigh level. After an interval of 24 hours the legs were reimplanted, using the nonsuture two-tube technic and vein grafts from a 3rd and 4th animal to bridge the defects in the femoral artery and vein. The amputated limbs were preserved during the 24-hour interval in cracked ice. The dogs were given sulfathiazole, one gram twice daily, by mouth, from the time of the first operation. The photographs were made 12 and 21 days, respectively, after reimplantation of the limbs, and there is every evidence of a good supply of arterial blood in these reimplanted legs. Correlation of these experiments with the informative studies of Brooks and Duncan[11] on the effects of temperature on the survival of anemic tissue is of interest.

The survival of the legs in these two dogs depended solely upon the function of vein segments transplanted from other dogs, and in this sense the experiments are critical. However, we have used heteroplastic vein grafts to bridge femoral artery defects in seven additional dogs. The anastomoses functioned for 17 days (average), which is well beyond the postulated 14 days of posttraumatic edema.

The present surgical trend of removing the great saphenous vein in ligating incompetent communicating veins has afforded us an opportunity of examining a number of these veins. We found that the average great saphenous vein is suitable for use as a vein graft for bridging arterial defects using our nonsuture method. The vein may be removed in lengths up to 40 cm., and is adaptable for use with vitallium tubes varying in diameter from 4–7 Mm. This means that the average great saphenous vein would be technically and functionally adequate for bridging defects in arteries varying in diameter from 4–12 Mm. This range of diameters would include the carotid artery or any artery one should care to anastomose in the extremities. The performance of our nonsuture method of anastomosis is not complicated by wide differences in diameter between the vein segment and the ends of the artery to be anastomosed. From a functional point of view, differences

in diameter up to 50 per cent are of relative little concern, for Mann[8] has shown that the lumen of an artery may be narrowed 50 per cent without affecting the volume of blood flow through the vessel. It is not until the lumen has been constricted in excess of 75 per cent that a sharp decline in volume flow occurs. What does occur, of course, at the site of a somewhat narrowed anastomosis, is a compensating increase in the rate of blood flow and this, in itself, is a protection against thrombosis at the site.

PRESERVED VEIN GRAFTS

We are now conducting experiments to determine the length of time that segments of veins may be preserved and function successfully when used to bridge arterial defects in dogs. Carrel[9] showed that segments of artery preserved up to five weeks in a refrigerator functioned indefinitely when used as heteroplastic grafts to bridge arterial defects in dogs. It is fortunate that in this war the equipment for preserving the segments of veins, the refrigerator, will be always fairly close at hand.

The most practical source of supply for veins suitable for use in bridging arterial defects in the war-wounded in active battle areas is *via* the pathologist. Segments of great saphenous, femoral, jugular or almost any sized veins may be removed aseptically by the pathologist. These veins may be placed in sterile test tubes and preserved in refrigerators against an occasion for emergency use. In less active battle areas, with the system of refrigeration it has been necessary to maintain for the transportation of blood plasma, it is not inconceivable to work out a plan for supplying great saphenous veins from civilian hospitals.

The one important thing to remember is that with a vein at hand, a severed carotid, or an artery in an extremity, may be quickly joined together by using the simple nonsuture method of blood vessel anastomosis.

SUMMARY

The problem of blood vessel anastomosis deserves important consideration in this war because (1) advances in the control of serious infection and the possible use of anticoagulants afford, for the first time in the history of wars, a basis for success; (2) the outstanding cause of the loss of limbs in this war will be damage to the blood supply.

We present vitallium as a nonirritating alloy suitable as a prosthesis for a vein graft bridging an artery or vein defect in a nonsuture method of vessel anastomosis using a single or double tube technic.

We have presented carefully controlled experiments on dogs demonstrating that sulfathiazole by mouth contributes greatly to the success of delayed anastomoses of severed vessels in contaminated wounds. Using sulfanilamide in alternate wounds in a series of 77 anastomoses we have noted it to be of moderate but definite value.[10]

The nonsuture method was shown to be highly successful in anastomosing

the small femoral arteries of dogs, even in contaminated wounds 24 hours after section of the artery, and without the use of anticoagulants.

We postulated that vein graft anastomoses of the severed primary artery in the war-wounded would prevent the loss of the extremity by gangrene if the anastomosis remained patent beyond the period of posttraumatic edema, up to 14 days.

Experiments are presented showing that veins taken from one dog may continue to function in another when used as transplants to bridge vessel defects. For example, the successful reimplantation of limbs 24 hours after their removal. These experiments afford a prospect for the use of preserved veins as grafts for bridging vessel defects in the war-wounded as an alternate to the use of homoplastic transplants.

CONCLUSIONS

If, as stated in the British War Memorandum, the indications for amputation are to be based primarily upon the integrity of the main blood vessels, a simple, quick method of joining vessels together should have much to commend it in this war. The control of serious infection, and the possible use of anticoagulants, would seem to insure success of the method.

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THE INTRACTABLE DUODENAL ULCER*

EVALUATION OF SURGICAL PROCEDURES

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DURING THE PAST DECADE even surgeons have admitted that medical management is the treatment of choice for duodenal ulcers and that surgical intervention should be confined to the intractable cases. The decision as to intractability varies greatly in different hospitals and clinics and the indications for operation which were used a generation ago are frequently used today, namely, obstruction, massive hemorrhage, a perforating ulcer, and the fact that the patient refuses to cooperate under medical management. It is perhaps because of the elasticity of indications for surgical intervention that gastro-enterologists question the advisability of any type of surgical procedure for duodenal ulcer.

My impressions, based upon 15 years experience in stomach clinics, lead me to question the above named indications for surgical intervention. On the Fourth Surgical Division of Bellevue Hospital we have observed 1,328 ulcers, representing 25,218 visits, and at Post-Graduate Hospital, since 1931, 676 ulcers have been seen, representing 7,628 visits. During the past ten years, in our experience, the only true indication of intractability in the case of a duodenal ulcer, has been constant pain. Relief from the continuous pain can not be obtained under any form of medical management, and sedation is frequently necessary to induce sleep.

Pyloric obstruction is still referred to in the literature as a true indication for surgical intervention. The stenosing and obstructing ulcer has recently been reviewed by Allen,[1] who states that the stenosing lesion "finally becomes a mechanical problem and the patient seeks help because he can no longer absorb nourishment to maintain life. The onset of such a condition is a gradual one, and the end-stage is reached so insidiously that one often finds that the stomach has become enormously dilated. It is interesting that such an organ emptying infinitesimal amounts of its contents into the duodenum, can exist within a person and create so little distress." In the two clinics with which I am associated, it has been our experience that a stenosing or obstructing ulcer, not accompanied by pain, does not require surgical intervention. These patients can be relieved by medical management and obstruction does not recur provided they remain free from pain.

Wilkinson[2] emphasizes the duration of the obstruction as the important factor. He states that an obstruction of three months duration, or less, will respond satisfactorily to medical management but in the case of an obstruction of three months, or longer, recurrences are almost certain and

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operation is usually indicated. He does not mention pain as a factor in causing the obstruction. In our experience, obstruction without pain is due to edema and pyloric spasm and not to scar tissue around the pylorus. Since 1932 no patient with so-called pyloric obstruction unaccompanied by severe pain, has been referred for surgical intervention, either in private consultation or in the clinics.

The exceptions to the above statement occurred in 1930 and 1931, when simple closure was done for perforated duodenal ulcers. Nine months after

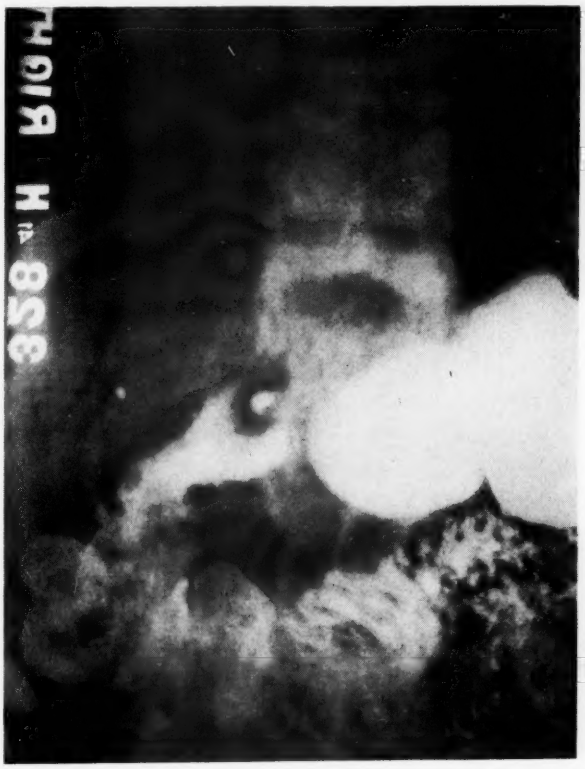

FIG. 1.—Penetrating posterior duodenal ulcer with symptoms of two months' duration. Operation advised by internist and roentgenologist.

operation in the case of one patient, and two years afterwards in the case of another, a true organic obstruction developed from the operative procedure, necessitating a short-circuiting operation. Neither of these patients was suffering from pain.

Massive hemorrhage, occurring once or oftener, as an indication for surgery brings up a controversial question. First of all, we do not have a clear-cut line between what constitutes massive and moderate hemorrhage, and a study of the literature reveals the elasticity in the grouping of these cases. However, if we all agree that a red blood cell count of 2,000,000 or less, a hemoglobin if less than 40 per cent, and lowered blood pressure with

a moderate degree of shock, constitutes massive hemorrhage, then we are obviously agreed that the patient has suffered a great loss of blood.

Surgical management of these patients may be considered under two headings: Surgical intervention in the stage of acute hemorrhage, which is occasionally indicated; and second, surgery as an elective procedure to prevent further hemorrhage. My views regarding the first mentioned type of management have been expressed in previous publications.[3, 4] The indications for surgery as an elective procedure are interpreted differently in different hospitals. The point emphasized by Allen[5] as to the dangers of massive hemorrhage in the different age-groups has been fairly universally accepted. That is, in a patient over 45 years of age, the prognosis is serious, while in a patient under 45, massive hemorrhage rarely proves fatal. Blackford and Cole[6] have confirmed this observation. Blackford and Allen,[7] in a report of 151 fatal hemorrhages, record that in only 35 cases, or 23 per cent, was there a history of previous hemorrhage, or in other words, 77 per cent of the fatalities occurred following the first hemorrhage—an interesting observation, with definite clinical significance. These authors also observed that 34, or 23 per cent of the deaths occurred at home. We have always felt, in considering massive hemorrhage from peptic ulcer, that unless patients are admitted under similar conditions the severity of the hemorrhage may be different. In a hospital with an active ambulance service the type of cases is different from those in the average hospital. The report of Blackford and Allen[7] indicates that, not infrequently, patients die at home before ever reaching a hospital. They state that fatalities in patients under 45 are rare, whether or not patients receive treatment, and that in patients under 40 fatalities are almost unknown. On this point, we must disagree. Since 1937, on the Fourth Division at Bellevue Hospital, I have had occasion to operate upon three patients who were medical failures, having had chronic ulcers for periods averaging from 5 to 15 years. These patients were suffering from intractable pain and were being prepared for operation on the surgical wards, when they had massive hemorrhages. Amazingly enough, all of them occurred within 24 to 48 hours of the day set for the elective operation. This group of cases included two duodenal ulcers perforating into the pancreas, and one gastrojejunal ulcer eroding into the transverse mesocolon and the pancreas. All three patients were saved by operative intervention. Massive blood transfusions were given before, during and after operation, and a subtotal resection was performed in each case. The patients were 27, 37, and 42 years old, respectively. Two patients were admitted as emergencies, with whom we had had no previous contact. One of them, 44 years of age, had a duodenal ulcer and the other, 33 years old, had a gastric ulcer. A subtotal resection was performed in each instance. The former patient died, the latter lived. There has been one other case in our division, a gastric lesion that resulted in a fatality while the author was out of town, but this case was proved at autopsy to be not an ulcer but a small ulcerating carcinoma.

From 1928 to 1937 we encountered an average of 13 massive hemorrhages a year, according to the definition of massive hemorrhage as previously described, and there was a medical mortality of ten per cent, while during the past six years, 1937 to 1942, inclusive, with surgical intervention in the desperate cases which do not respond to continuous transfusions, we have lost but one case from massive hemorrhage after surgical intervention, and none under medical management.

The penetrating or perforating duodenal ulcer, as diagnosed roentgenologically, does not necessarily constitute a true indication for surgical inter-

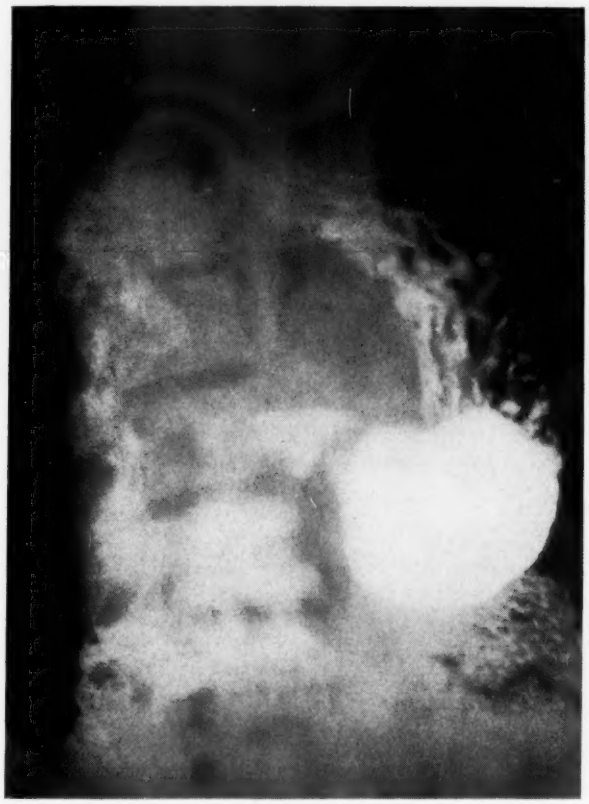

FIG. 2.—After three weeks' observation on the surgical wards.

vention. Here, again, one must rely on the clinical evaluation of the symptomatology. If the patient has had an ulcer for years, with intractable pain, then the indications are obvious, but if he has had an ulcer for only a short period of time, which proves to be a penetrating ulcer, medical management will very frequently relieve the symptoms and the niche will disappear.

The following case was referred to me for operation in January, 1941. The patient was a young man, 24 years of age, with a history of ulcer of two months duration. Roentgenograms revealed a penetrating posterior duodenal ulcer (Fig. 1), and the internist advised operation. After seeing this patient

in the hospital I advised against operation, and recommended medical management, and after three weeks the niche disappeared, the symptoms were relieved, and the patient has remained well since that time (Fig. 2). The indication, therefore, for surgical intervention in the penetrating ulcer can be decided only by the clinical course, and not by the roentgenologic appearance of the lesion.

The patients who will not cooperate under medical management should not be operated upon. If one accepts this type of case as an indication for operation the results from any type of surgical procedure, whether it be a gastro-enterostomy or a subtotal resection, will be most unsatisfactory.

There is a great difference of opinion as to why we obtain good results in any surgical procedure for a duodenal ulcer. Perhaps the most generally accepted opinion is that it is due to a reduction in the gastric acidity, whether the operative procedure has been a gastro-enterostomy or a subtotal resection. We started 15 years ago to observe patients treated both medically and surgically, and to evaluate the reasons for failure in each group. We soon realized that a gastro-enterostomy undertaken for the adequately treated cases was very unsatisfactory. In our clinics at Bellevue Hospital we found that patients whom we were referring for gastro-enterostomy 12 to 15 years ago, constituting the intractable case with severe pain, obtained very poor results from surgery. The author personally had performed 29 gastro-enterostomies for duodenal ulcer in this group, and was amazed at the high percentage of failures from the short-circuiting procedure, so it was abandoned completely after 1932.

It is difficult, if not impossible, to evaluate the pathologic process in a duodenal ulcer unless a subtotal resection is done, and in the 29 gastro-enterostomies the ulcer was described as anterior in 14 instances, posterior in 13, and anterior and posterior in two instances. That is interesting when one evaluates 75 subtotal resections performed for duodenal ulcers in the same clinic, in which group there were 80 per cent posterior wall ulcers, with a definite chronic pancreatitis, in ten instances, or 13 per cent, there was both an anterior and a posterior wall ulcer, with pancreatitis, and in only five, or seven per cent, was there a single anterior wall ulcer. The ages of the patients in the two series were identical—37.1 per cent for the gastro-enterostomies, and 37.1 per cent for the subtotal resections. The symptoms in the gastro-enterostomies had persisted for 5.4 years and in the subtotal resections 5.3 years, which would indicate that we were treating, in all probability, identical ulcers.

The exact location of the duodenal ulcer is impossible to determine unless a subtotal resection has been performed. This point is illustrated in Figures 3, 4, 5 and 6.

Our opinion as to the best surgical procedure for intractable duodenal ulcer has been arrived at by evaluation of the results obtained from gastro-enterostomies performed in the stomach clinic of the Fourth Division of

Bellevue Hospital, and reported[8] in 1934, and again[9] in 1940. From these reports conclusive evidence was produced to show that gastro-enterostomy was a most unsatisfactory method of treatment. In 1934 we reported 96 gastro-enterostomies, followed for an average of 4.2 years, with 37 per cent cures and 16.7 per cent gastrojejunal ulcers. In 1940 we reported 106 gastro-enterostomies, followed for an average of 7.1 years, with 24.5 per cent cures and 18.8 per cent gastrojejunal ulcers.

Since 1933 we have performed subtotal resections, exclusively, in all cases of duodenal ulcer. I am willing to admit that there is one indication for a gastro-enterostomy for duodenal ulcer, namely, the stenosing lesion which results from operative intervention for a previous acute perforation not associated with pain. Then a short-circuiting procedure should prove satis-

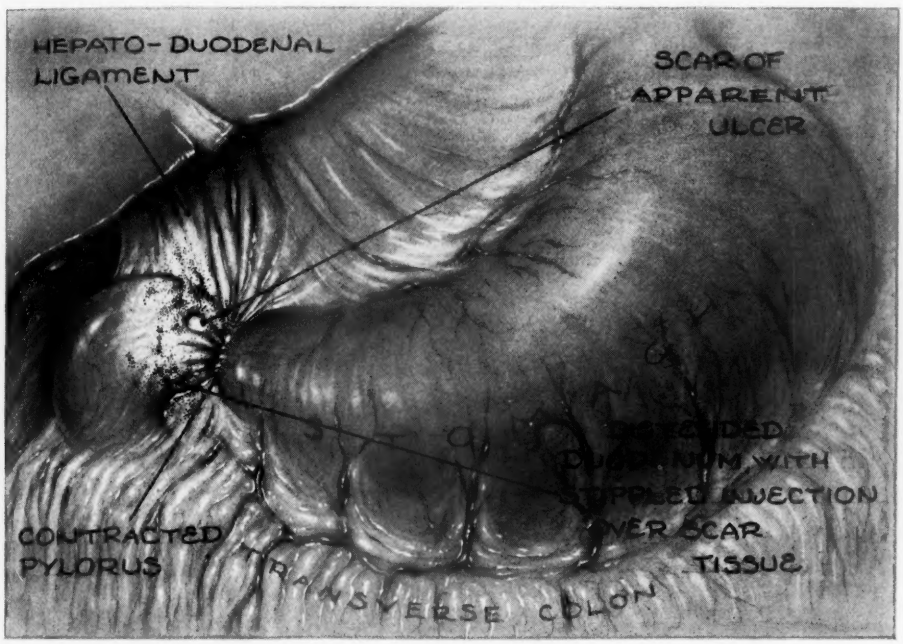

FIG. 3.—Drawing from an apparent anterior duodenal ulcer, but see Figure 4.

factory, but during the past ten years we have not encouraged such a case in either of the clinics with which I am associated, or in patients seen in private practice.

Subtotal resection is now accepted as the operative procedure for intractable duodenal ulcer. The term subtotal resection is rather loosely used, according to all recent writings, but the generally accepted concept merely means removal of one-half to three-fourths of the stomach, leaving *in situ*, if advisable, the pyloric end of the stomach and the duodenum with the adherent ulcer to the pancreas. Those who advocate leaving the adherent ulcer and even the pyloric end of the stomach believe that the good results are based to a great extent, if not entirely, upon achlorhydria resulting from

subtotal resections. Little stress is placed upon ridding the patient of the inflammatory mass, which would seem to be essential in obtaining a good result.

Colp[10] reported 502 subtotal resections, with 40 recurrences, and in 33 of the 40 the ulcer had been removed. He is definitely of the opinion that the removal of the ulcer has little to do with the recurrence and he goes on to state that the incidence of recurrence will be minimal in cases of anacidity but if free acid is present after operation the incidence of recurrence is going to be much greater. From my observations of patients in our clinics and in private practice, where the ulcer has been left *in situ* by other surgeons, there are more unsatisfactory results than in cases in which the ulcer has been removed *in toto*. A recent report[11] from our clinic at Bellevue Hospital shows that in none of the cases operated upon on our service, who have been followed, was a gastrojejunal ulcer found. In this group we had 104 subtotal resections followed on an average of 2.88 years, and these patients had made 1,822 visits, or 18 visits per patient. No case was reported which had not been followed for one year or longer. Sixty-five per cent of these patients were cured, which means they were completely symptom-free. Twenty-five per cent were improved, and the chief reason for placing them in this group was weight loss, without pain. The weight records in all cases of subtotal resections are interesting. In 47.6 per cent there was a weight loss, in 39.2 per cent the weight remained unchanged, and 8.4 per cent they lost and then gained weight.

If one accepts the teaching that achlorhydria is the goal to reach in subtotal resection, then there is a definite reason, in all instances, for removing the pylorus and the adherent ulcer. Experimental evidence shows that the mucosa of the pyloric end of the stomach affects the secretion from the cardia and fundus of the stomach by the hormone "gastrin," as shown by Edkins,[12] and Koch, Luckhard and Keston.[13] These authors believe that the hormone is produced and thrown into the blood stream by the pyloric mucosa. Because of the similar action of histamine upon the gastric mucosa, several authors believe this hormone to be histamine. However, recent work on isolated and denervated gastric pouches with histamine-free extracts demonstrated the true hormone character of the internal secretion of the gastric mucosa.[14, 15, 16]

There is little doubt that in those cases in which a subtotal resection of 50 to 75 per cent of the stomach is done and the duodenal ulcer is left *in situ*, the follow-up results are far from satisfactory. This point has been stressed by Friedell, Shaar and Walters,[17] in which they state "while the prognosis as to ultimate cure and certainly as to the immediate relief is favorable, it surely does not present the same chance of a complete cure that pyloric removal does," and they cite a case in which the pyloric portion of the stomach and the duodenal region was not removed, but the gastric mucosa was dissected from the remaining pyloric stump. Analysis of the gastric con-

tents three weeks after operation revealed 10 to 15 units of free hydrochloric acid in the gastric section which increased to 60 units after the administration of 0.5 mg. of histamine. They believed that neglecting to remove the pyloric portion of the stomach was a factor in the failure.

Kiefer[18] has elaborated upon this same point in reporting 222 subtotal resections for peptic ulcer. He emphasizes that 49 were for gastric lesion and 173 for duodenal ulcer. "In 30 cases, because of technical difficulties and their attendant risks, the duodenum and pylorus were not included in

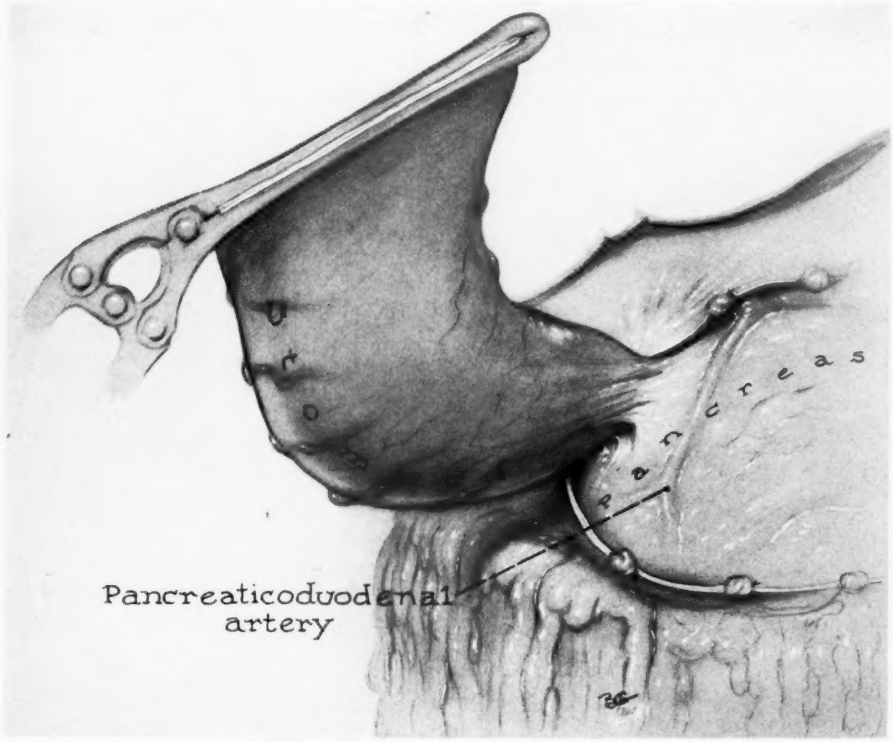

FIG. 4.—Ulcer actually posterior and perforating into head of pancreas.

the resection, the distal transection being placed proximal to the pylorus, as recommended by Finisterer in cases in which removal of the duodenum is too dangerous. In this group there were seven recurrent ulcers and three cases of postoperative hemorrhage. This relatively high incidence of unsatisfactory results has led to speculation regarding the rôle of the pylorus and the antrum since there is evidence that a hormone is evolved in the pars pylorica which stimulates the fundic glands to produce acid. However, actual evidence indicating that failure to remove the pyloric end of the stomach was solely responsible for the recurrence of the ulcer is rather meager. A post-operative gastric analysis after an Ewald meal was done in 24 cases of this group with the finding of free acid in 13 cases, which is approximately the same proportion as that found in the combined group

of duodenal ulcer cases. The fact that the surgeon had decided against the attempt to remove the pylorus was an indication that this group represented a particularly severe grade of peptic ulcer and might be expected to show a higher percentage of recurrence."

Whether or not a hormone is secreted by the pyloric end of the stomach, there seems to be little doubt, in some of the clinics with large experience with subtotal resections, that in those cases in which the duodenal ulcer is left *in situ* the results are not as satisfactory as when it is removed. The two previous reports vary as to their interpretation. One states that the result is due to increasing incidence of the acid; the other that it is due to a more virulent type of ulcerative process. If the latter opinion is correct, then there is every reason for removing such a lesion.

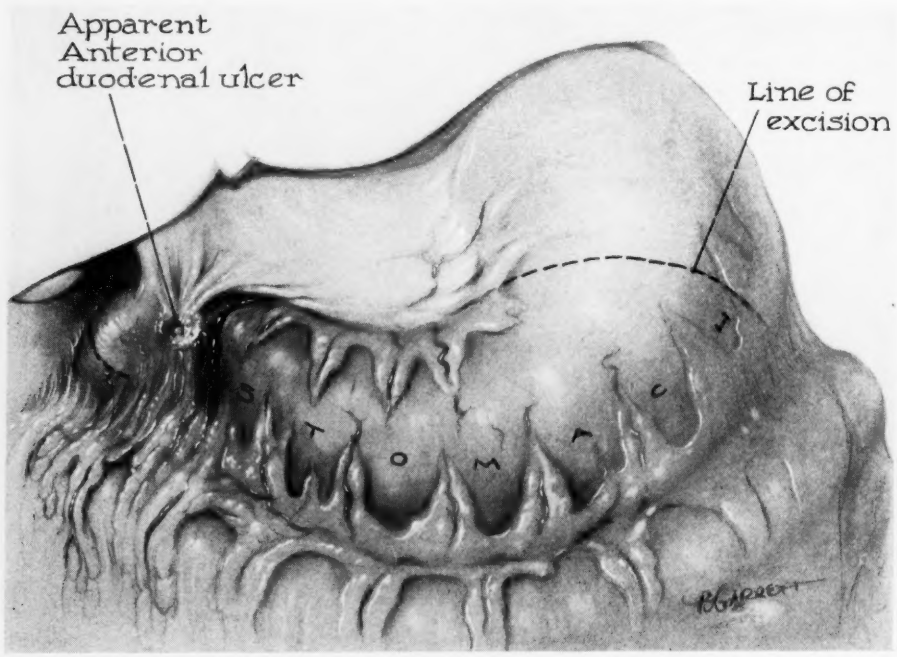

FIG. 5.—Apparent anterior duodenal ulcer, but see Figure 6.

TECHNIC OF SUBTOTAL RESECTION

During the past ten years in every operation for duodenal ulcer, I have removed 65–70 per cent of the stomach, and the pylorus and the duodenal lesion *in toto*. One is constantly seeing in the literature statements to the effect that the lesion could not be removed due to the technical difficulties encountered. From my experience during the past ten years, which, in the clinics and private practice, includes over 150 cases, no case has been encountered in which the ulcer could not be removed. One of the very interesting phases of the so-called difficult lesion is the relative ease with which the apparently difficult ulcers were removed. Some of the smaller lesions are often much more difficult to remove technically than the large

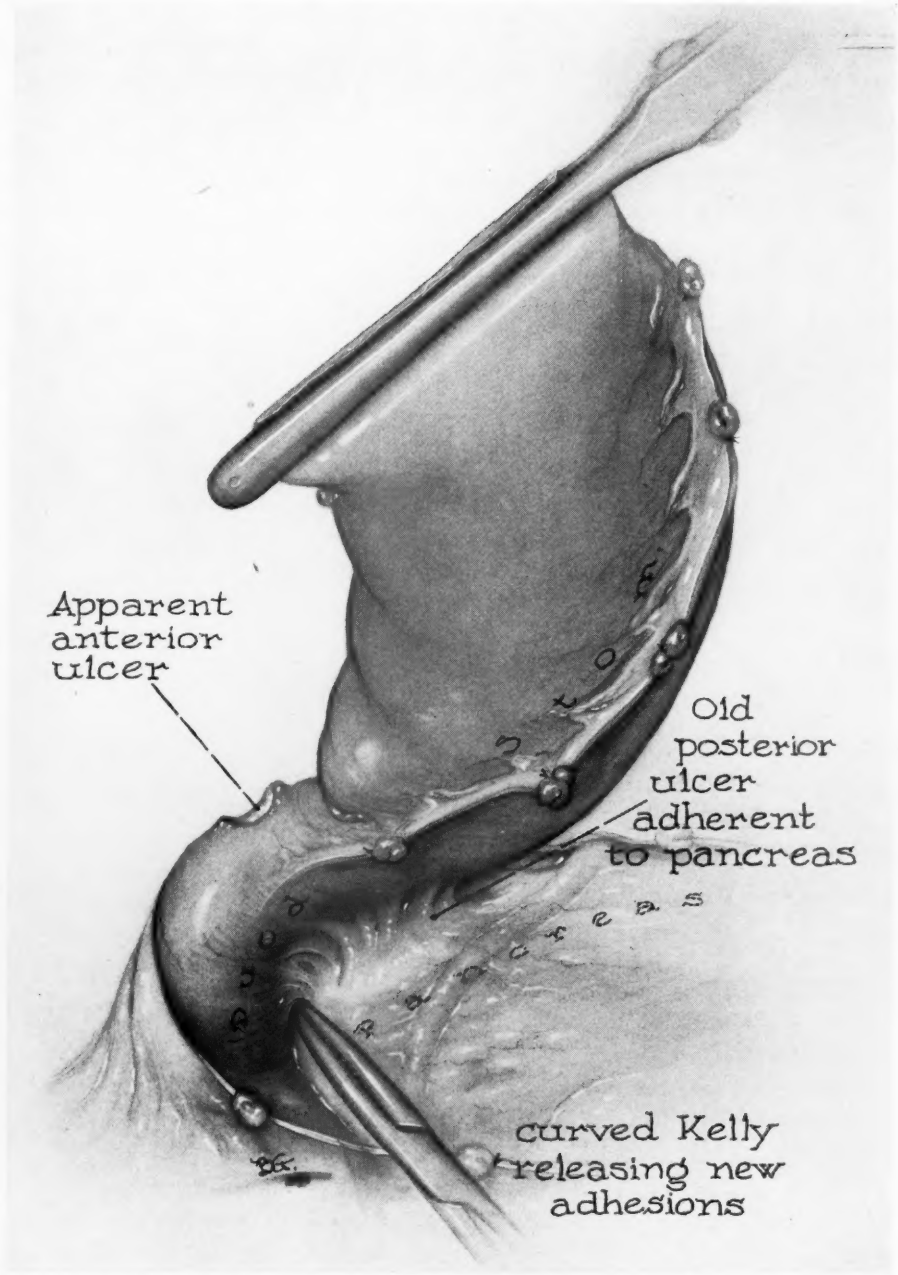

FIG. 6.—Ulcer posterior and perforating into head of pancreas.

inflammatory mass. The inflammatory process has completely occluded all the smaller vessels and the process is similar to removing the acutely inflamed gallbladder as compared with one subacutely or chronically inflamed. The former operation is technically much easier to perform. The same treat-

ment applies to the large inflammatory masses around the pancreas and duodenohepatic ligament.

The technic followed is to divide the gastrocolic omentum just proximal to the adherent mass and carry this proximally along the greater curvature to a point well above the reentrant angle of the stomach, after which the vessels in the gastrohepatic omentum are divided and then the stomach transected from right to left above the reentrant angle. This procedure provides an opportunity for exposing the adherent mass in the pancreas and duodenohepatic ligament and for freeing it under better visualization. After

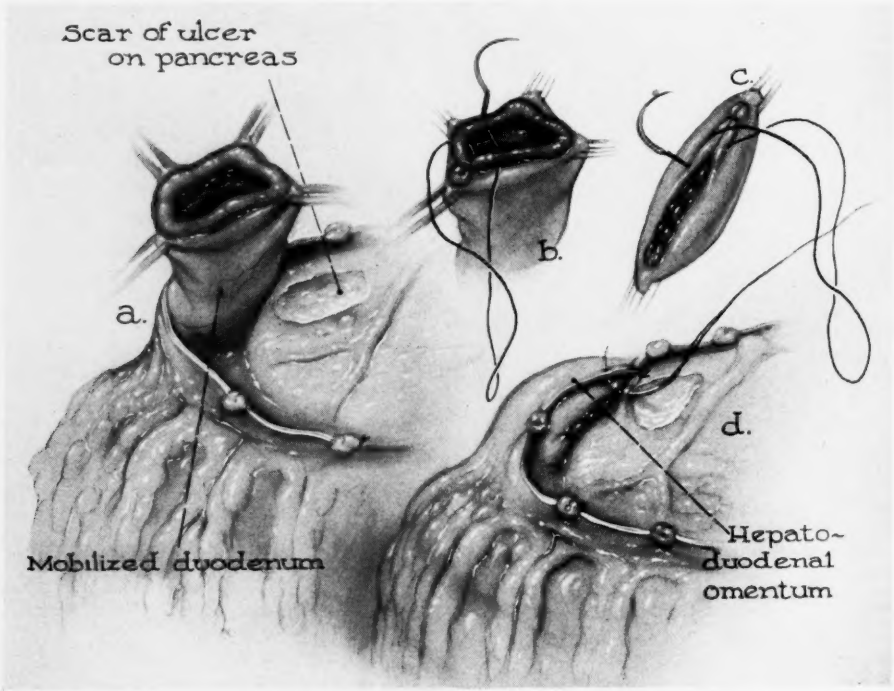

FIG. 7.—The duodenal ulcer can always be removed and the stump closed.

the inflammatory mass has been completely separated, the duodenal stump is carefully closed with cotton sutures using four layers to completely invert the stump and bury it in the head of the pancreas in the region of the previous perforation (Fig. 7). The most important part of the whole operative procedure, as it affects mortality, is the closure of the duodenal stump. After this the continuity between stomach and jejunum is reestablished, as illustrated in Figure 8. Three layers of cotton sutures are used posteriorly and three layers anteriorly. The abdomen is always closed, without drainage, by using interrupted cotton sutures for the peritoneum, the fascia and the skin.

The preoperative care of ulcer patients is an important factor in their surgical management. In a previous report,[19] pre- and postoperative physiologic balance was discussed, and the course of those patients operated upon on the Fourth Surgical Division at Bellevue Hospital evaluated. These

patients averaged 17 days in the hospital preoperatively, and 19 days postoperatively. Although the majority had been under observation for years before operation was decided upon, this period gave us added time to further evaluate the severity of their pain and to completely restore their fluid balance

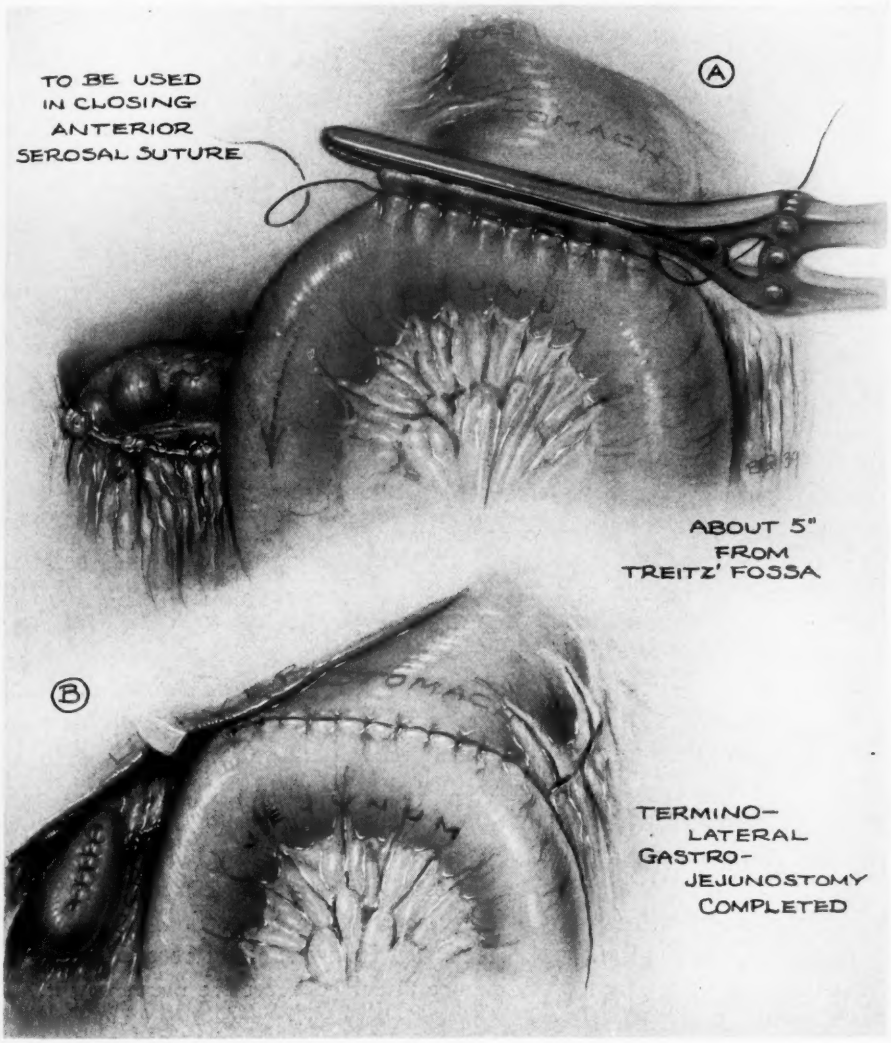

FIG. 8.—Method of performing an anterior short-loop anastomosis without entero-enterostomy.

and vitamin B and C deficiencies. They were always given 2,000 cc. of saline with 500 mg. of vitamin C and 100 mg. of vitamin B once a day for seven days preceding operation. Saline is used in preference to glucose, as the fluid remains in the tissues and does not have a diuretic action.

The type of anesthesia used has always been endotracheal, and the choice of the agent, whether it be cyclopropane or ether, or a combination of the two, is always left to the discretion of the anesthetist. Anesthesia in gastric

surgery has recently been reviewed by us.[20] Spinal anesthesia has not been used in any of the resections during the past ten years.

The postoperative management of these patients is important. Morphine should be used very sparingly. Large doses depress the respiratory tract, and, as Waters[21] has aptly put it, "the cough reflex is eliminated which is the janitor of the respiratory tract." The patients are turned from side-to-side every hour. They are given an average of 5 mg. Magendi solution preoperatively and 15 mg. postoperatively. The Levine tube is passed one hour before operation, it is irrigated every hour postoperatively for 48 hours and left open for gravity drainage. Fluid is given by mouth as soon as the patient reacts, as it serves as a cleansing agent for gastric mucin. Postoperatively, the intravenous administration of fluid should be carefully watched as these patients are well hydrated preoperatively. During the first 24 hours, postoperatively, they should receive not more than 2,000 cc., and after 48 to 72 hours, intravenous administration of fluids may be discontinued.

In a previous report[22] of operations at Bellevue Hospital, by the author, there were 102 subtotal resections for ulcer, with five deaths, one of which was attributed to pneumonia and the other four to peritonitis. Of the 102 patients, 33 had previously undergone gastric surgery. These cases, of course, are from a municipal hospital, without special nurses, and with all the other handicaps under which one works in taking care of major surgical cases in such an institution.

CONCLUSIONS

1. From my personal observation of a large number of chronic duodenal ulcers, I believe that the only indication for operation is uncontrollable pain which does not respond to any form of medical management.

2. When these patients are submitted to surgery, there is only one operative procedure which seems justifiable, namely, a subtotal gastrectomy, with removal of the pylorus and the ulcer *in toto*.

3. If the indications given above have been observed, gastrojejunal ulcer is a most uncommon occurrence following a subtotal resection.

4. Gastrojejunal ulcers will, of course, follow subtotal resections unless the patient has been given every chance under conservative management.

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NITROGEN METABOLISM, CALORIC INTAKE AND WEIGHT LOSS IN POSTOPERATIVE CONVALESCENCE*

A STUDY OF EIGHT PATIENTS UNDERGOING PARTIAL GASTRECTOMY FOR DUODENAL ULCERS

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THE SCIENCE OF NUTRITION has not until recently engaged the serious attention of the surgeon. This is attributable to several factors. The nausea and vomiting, the impairment of appetite and the derangement of digestive processes caused by anesthesia, and the pain and the emotional stress connected with an operation, and by the operation itself, are not conducive to healthy alimentation. Added to this may be mentioned the fear on the part of the surgeon of placing food into a viscus which has been the site of a recent operation. However, since the usual surgical case requires dietetic restriction only for a few days postoperatively, and since most patients come to operation with enough body stores to tide them over this lean period, the surgeon has rarely been impelled to regard nutrition as a matter of any urgency.

The work on the rôle of hypoproteinemia in surgical conditions by Jones, and his coworkers,[1] Ravdin, and his associates,[2, 3] Koster and Shapiro,[4] and Hartzell, and his coworkers,[5] the study by Cuthbertson,[6] in England, of nitrogen loss, sometimes called the "toxic loss of nitrogen," and by Elman,[7] and Brunschwig, *et al.*,[8] in this country, and the demonstration by these last two workers of the safety and feasibility of intravenous alimentation, constitute a new chapter in surgery. The work of these men has, at last, linked surgery with the basic work of pioneers like Van Slyke, Rose, Whipple, Madden, Weech and others.

While it is true that most surgical patients need little nutritional attention, there are surgical conditions in which the maintenance of the nutritional state is a matter of grave importance. One of these conditions is peptic ulcers. Thus, Riggs, Reinhold, Boles and Shore[9] found statistically significant deficiencies in serum total protein, albumin and vitamin C concentrations in a group of 52 cases of peptic ulcer. Many of these ulcer patients, either as a result of dieting or of repeated bleeding, have lost considerable weight, with the blood protein at or below the lower limit of normal, and the body stores well depleted. Some lend themselves to preoperative "building up,"

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but others may require emergency operations while in their depleted state. If a major operation, such as gastrectomy, is performed upon these patients, there is added to the "toxic loss of nitrogen" the partial starvation imposed by a highly restricted postoperative regimen, which further depletes the body tissues, interferes with healing, and may lead to wound dehiscence. An example of this is D. R., reported in Table I.

In the present study the nitrogen balance was determined in eight cases of duodenal ulcers who had undergone partial gastrectomy. They were divided into two groups: four cases who were given the postoperative ward routine of infusions, occasional blood transfusions, and a gradually increasing oral feeding. This may be called the control group. In the other group of four cases, a high caloric and high nitrogen feeding was given to replace or more than replace the nitrogen loss, so that at the end of the postoperative period of 10 to 12 days, a nitrogen surplus was accumulated in the body. In addition to the nitrogen balance, both the body weight and the plasma proteins in both groups were determined periodically throughout the convalescent period. The fluid intake and output were followed and the caloric and sodium chloride intakes were noted. The chief sources of nitrogen loss were recorded. Also noted, but not objectively studied, was the "strength" of the patient. The number of days of hospitalization of one group was compared with that in the other.

Tables I to IV each represents a control case; Tables V to VIII each a high nitrogen feeding case. Graphs a, b, c and d in Figure 1 are records of the control group, and corresponding graphs in Figure 2, of the feeding group, showing the important representative findings.

EXPERIMENTAL CONSIDERATIONS

Preoperative Preparation of the Patient.—The patients in the control group each had a Levin tube introduced through one of the nares into the stomach for decompression of this viscus by the Wangensteen suction apparatus during the first four to six postoperative days. In the feeding group the Levin tube was replaced by a double-lumened tube, adopted from a Miller-Abbott tube in such a way that one barrel was longer than the other by some ten inches. The shorter barrel was for suction and the longer for feeding. The ends of both barrels were perforated at several levels, in order to facilitate feeding and suction. During the operation, after the stomach was resected, and while the anastomosis was being made, the feeding barrel was introduced through the stoma into the jejunum, after which the operative routine was resumed. Immediately postoperatively, Wangensteen suction was made through the suction barrel, which lay in the stomach.

The Weight of the Patient.—The weights of the patients were taken on the Howe platform scales. In the case of preoperative weights it was a simple matter for the patients simply stood on the scale platform for the weighing. In the postoperative period, however, the matter of weighing was somewhat more complicated. If the patient was light: *i.e.*, not over 60

Kg., he was lifted by an orderly and the weights of both the orderly and the patient were taken, after which the weight of the orderly was then subtracted from the combined weight. If he was over 60 Kg., he was placed on a stretcher and the loaded stretcher was then weighed on two scales, the two front wheels on the platform of one scale and the hind wheels over that of another. The two scales used in this work were sensitive to within 25 Gm., so that where two scales were used, the sensitivity would be decreased by half, and the margin of error would be in the neighborhood of 50 Gm. Since it was not the absolute weight but the comparative readings taken over several weighings for the construction of the weight curve, which was important, this margin of error was not serious.

The Fluid Intake.—In the control group, the fluid intake was maintained mainly by intravenous infusions of 5 or 10% dextrose solutions in distilled water or in physiologic saline, some for the first four or five days and some for a longer period. Occasionally amigen solution was also given intravenously. The amount lost by gastric suction was replaced by a corresponding amount of saline, according to Coller and Maddock's[10] principle. This accounts for the large fluid and NaCl intake of the first four to six days in both groups. After the first four or five days, when fluid intake by mouth was being gradually increased, the infusions were tapered off correspondingly.

In the feeding group, the first two cases (A. V., Table V and F. R., Table VI) had a large number of intravenous infusions. In the last two cases, however, infusions were given only in the first postoperative 12 hours and thereafter the fluid intake was entirely given through the feeding tube or by mouth.

The Chloride Intake.—An attempt was made not to exceed the chloride intake of nine grams daily. However, the problem was complicated by the loss of fluid through the gastric suction. As stated above, the Coller and Maddock[10] principle required this amount to be replaced quantitatively in physiologic saline, and this accounts for the apparently unduly large amount of sodium chloride the first four to six days. However, a possible source of error is present in this volume-for-volume replacement. If the suction were continued while the patient took a drink of water, the drainage might contain less sodium chloride than was assumed in the Coller and Maddock principle and, consequently, the patient would be given more sodium chloride than he needed. In order to obviate this, the suction was turned off for half an hour after each drink. Whether in this time the water had all passed out of the stomach or become isosmotic with systemic fluids is not known.

The Caloric and Nitrogen Intakes.—In the control group, the caloric intake was derived from three sources: (a) Infusion of dextrose solutions, four calories being assigned to each gram of dextrose; (b) peptonised milk and the usual articles of soft diet, the caloric and nitrogen values of which were taken from the dietitians' chart; and (c) the few calories and small amount of nitrogen derived from the plasma proteins in the blood trans-

TABLE I

CASE OF D. R., MALE, AGE 50

Date	Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Chloride Intake Gm.	Cl. Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N Status Gm.	P.P. Gm. % A/G	Wt. Kg.	Remarks
March 8													3.½.4	63.4	
9 to 10	3250	980	1300	20.25	870	2.8	7.983	.809	.593	9.466	− 6.666	− 6.666			Tr.—500 cc. W.B., Infusion; 2L. —10% dex.; 1L. saline.
10 to 11	3000	750	1200	18	1140	5.6	15.877	.771	.593	17.241	−11.641	−18.307			Tr.—500 cc. plasma—Inf.: 1L. 10% dex. in D/W; 1½ L. in saline.
11 to 12	3000	860	1000	9	800	0	10.995	5.656	.593	−17.244	−17.244	−35.551			Inf.: 1L. 10% dex. in D/W; 1L. in saline—1000 cc. H_2O by mouth.
12 to 13	3000	960	1050	9	800	0	6.227	7.665	.593	14.485	−14.485	−50.036	5.4	61.25	Inf.: Same as above.
13 to 14	3250	920	1240	18	800	0	7.583	1.297	.593	9.473	− 9.473	−59.509			Inf.: 2L. 10% dex. in saline. 1250 cc. H_2O by mouth.
14 to 15	3500	1050	1100	18	800	0	8.142	2.5	.593	11.235	−11.235	−70.744		59.15	Inf. as above. H_2O by mouth to volume.
15 to 06	2500	700	850	9	940	5.6	17.028	3.559	.593	21.18	−15.58	−86.324	5.25		Evisceration discovered 16th; re-sutured. Inf.: 2L. 10% dex. 1 in saline, 1 in D/W; Tr. 500 cc. plasma.
16 to 17	2250	0	1150	9	870	2.8	13.354	—	.931	14.285	−11.485	−97.809			500 cc. W.B. 1L. 105% dex. in D/W; 1L. in saline.
17 to 18	3820	—	1342	9	1231	6.97	6.886	—	.931	7.817	− .847	−98.656			2L. 10% dex. in D/W; Inf. Pep. milk by mouth 6 glasses; 500 cc. H_2O.
18 to 19	2200	—	855	9	1342	6.342	14.115	—	.931	15.046	− 8.704	−107.360	5.32		1L. 10% dex. in D/W; 1200 cc. Pep. milk. No manifest edema
19 to 20	3000	—	1107	11.88	1342	6.342	12.142	—	.931	13.073	− 6.731	−114.091	5.20	56.45	6 glasses milk + water. Wound shows no signs of healing.
Total		6220				36.454	120.332	22.25	7.875	−150.545					
Average		887				3.314	10.939	3.18	.716	− 13.686					
20—on soft diet + 500 cc. nutramigen.															
4—2													6.17	61	Wound begins healing March 24.
April 13													6.65	62.5	Discharged. Wound partially healed.

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TABLE II

CASE R. B., MALE, AGE 33

Date	Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N Status Gm.	P.P. Gm. % A/G	Wt. Kg.	Remarks
May 11													6.15	64.32	
12 to 13	3250	450	1700	13.5	670	2.8	11.909	.841	.351	13.106	−10.306	−10.306			500 Tr. W.B. Inf. 1L. 5% dex. in saline; 2L. in D/W.
13 to 14	3850	920	1725	16.65	1468	6.92	16.917	1.113	.351	18.381	−11.461	−21.767			Tr. 350 cc. plasma. Inf. 500 cc. 5% amigen in 5% dex.; 2L. 10% dex. in D/W; 1L. ditto in saline.
14 to 15	3500	450	2200	13.5	1340	5.6	14.9	.330	.351	15.581	− 9.981	−31.748	5.64	61.6	Tr. 500 cc. W.B.; 250 cc. plasma. Inf. 2L. 10% dex. in D/W; 1L. in saline.
15 to 16	3250	1800	2000	11.25	870	5.6	16.812	1.975	.351	19.138	−13.538	−45.286			Tr. 500 W.B.; 1L. 10% dex. in saline. 2L. 5% dex. in D/W.
16 to 17	3250	0	2200	11.25	1210	8.8	17.757	—	.351	18.108	− 9.308	−54.594			Tr. 250 cc. plasma; Inf. 1000 cc. 5% amigen; 2L. 10% dex. in D/W.
17 to 18	2690	0	1800	13.25	1154	11.123	16.757	—	.351	17.108	− 5.985	−60.579		60.68	Tr. 250 cc. plasma; Inf. 2L. 5% amigen in 5% dex. 1L. 10% dex. in D/W. 440 cc. pep. milk.
18 to 19	2660	0	1500	5.94	1271	3.489	15.64	—	.351	15.991	−12.502	−73.081			Inf.—2L. 10% dex. in D/W.
19 to 20	1880	0	1650	7.92	692	4.646	13.42	—	.351	13.771	− 9.125	−82.206	5.52	60.00	4 cups tea with 1 teaspoonful sugar each. 880 cc. pep. milk.
Total		3620				48.978	124.212	3.259	2.808	131.184					
Average		905				6.123	15.462	.815		16.398					
20—on Sippy diet + 500 Gm. nutramigen.															
June 3													7.01	64.3	
June 5														64.8	Discharged.

TABLE III

CASE F. W. MALE, AGE 50

Date	Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Nacl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N. Status Gm.	P.P. Gm. % A	P.P. Gm. % G	P.P. Gm. % Hem.	Wt. Kg.	Remarks
Dec. 13																57.97	
14 to 15	4250	880	1650	18	1010	14.8	11.944	.642	.692	13.278	+ 1.522	+ 1.522	4.1	2.4	25		Tr. 500 cc. W.B. Inf. 2L. 5% agimen and 5% dex. 1L. 5% dex. in 1L. saline.
15 to 16	3660	450	1500	9	600	0	10.964	.135	.692	11.791	−11.791	−10.269	5.5		32	57.71	2L. 5% dex. in saline. 1L. 5% dex. in H_2O; H_2O by tube = 660 cc.
16 to 17	2050	400	1500	0	80	0		.150	.692								600 cc. tea with 80 Gm. sugar.
17 to 18	2050	440	3550	0	80	0	25.886	.137	.692	26.173	−22.156	−32.425					1450 cc. H_2O.
18 to 19	2050		1230	0	700	4.017			.692				5.35		31	55.68	Ditto.
19 to 20	?		875	?	850				.692								760 cc. pep. milk + H_2O + broth.
20 to 21			1000		850	17.123	23.754		.692	25.830	− 8.707	−41.132				55.68	1080 cc. pep. milk + broth + H_2O.
21 to 22			1375		850				.692								Ditto.
22 to 23			1250		1130				.692				7.04		38		Ditto.
23 to 24			700		1900?	23.67	24.639		.692	26.715	− 3.045	−44.177					Ditto.
24 to 25			1400		1900?				.692								Soft diet (partially consumed).
25 to 26			1350		1900	6.5	6.429		.692	7.121	− .621	−44.798				54.54	Soft diet (partially consumed).
28													6.8		39	53.52	
Total		2170				66.110	126.16	1.064	8.304	110.908							
Average		543				5.509	18.025	.216	.692	9.242							
Jan. 3																	Discharged.

TABLE IV

CASE J. S., FEMALE, AGE 48

Date	Fluid Output: Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl. Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N Status Gm.	P.P. Gm. % A/G	Hem.	Wt. Kg.	Remarks
Dec. 9													7.48	43	85.23	
10 to 11	2500	1000	830	45	940	5.6	7.558	4.67	.22	12.448	− 6.848	− 6.848				In 1L. W.B.; 2L. 10% dex. in D/W.
11 to 12	3300	2650 (bloody)	725	9	1200	0	6.886	25.368	.22	22.474	−22.474	− 29.322				Inf. 3L. 10% dex. in D/W; 300 cc. H_2O by tube.
12 to 13	3050	1600 (bloody)	930	18	800											300 cc. H_2O by tube. Inf. 2L. 10% dex. in H_2O; 600 cc. H_2O + 450 cc. tea.
13 to 14	3150	2650	850	9	600	0	37.58	39.368	.659	83.538	−83.538	−112.86				Inf. 1L. 5% dex. in saline; 1L. 10% dex. in H_2O; 1150 cc. H_2O and tea.
14 to 15	2800	1650	680	9	400										81.81	Inf. 2L. 5% dex. in H_2O; 1L. in saline· 800 cc. H_2O and tea.
15 to 16	?	—	950	?				0					6.34	38		4 glasses pep. milk; water and tea to 2500 cc.
16 to 17	?	--	1200	?	2220	14.27	41.87	0	.659	42.529	−28.259	−141.119				5 glasses pep. milk; H_2O and tea.
17 to 18	?	—	1100	?	?			0					6.12	37	80.00	4½ glasses pep. milk; H_2O and tea.
Total		9550				19.87	93.894	69.406	1.758	160.989						
Average		1950				3.974	11.737	17.451	.21	20.125						
18—On soft diet.																
21															81.36	
27													6.54	38	81.36	
Dec. 27																Discharged.

TABLE V

CASE A. V., MALE, AGE 47

Date	Fluid Output: Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	N. Deficit or Surplus	P.P. Gm. % A/G	Hem.	Wt. Kg.	Remarks
Mar. 31													6.57	69.77		
April																
1 to 2	2250	825	650	11.25	470	2.8	3.764	2.100	.965	6.829	−4.029	− 4.029				Tr. 500 cc. W.B. 1L. 5% dex. in saline; 1L. ditto in D/W.
2 to 3	3800	1500	1050	13.50	4025	20.25	8.9	1.576	.965	11.441	+8.809	+ 4.78				750 Gm. nutramigen by mouth in 1500 cc. saline—water to make 3800 cc. Inf. 1L. 5% dex. in D/W.
3 to 4	4500	2600	700	18	4025	20.25	13.207	3.036	.965	17.208	+3.042	+ 7.82		69.8		Ditto—with 2000 cc. saline and H_2O to make 4500 cc.
4 to 5	4500	2800	1000	18	4025	20.25	10.6	2.615	.934	14.149	+6.101	+13.923	6.72			Ditto—with 2000 cc. saline and H_2O to make 4500 cc. Inf. 1L. 5% dex. in D/W.
5 to 6	4500	1700	1150	18	4025	20.25	10.787	1.667	.941	13.395	+6.855	+20.778		70.5		Ditto—with 2500 cc. saline and H_2O to make 4500 cc. Inf. 1L. 5% dex. in D/W.
6 to 7	3000		1900	18	4025	20.25	10.796		.941	11.737	+8.513	+29.291				Ditto—with 2000 cc. saline and H_2O to make 3000 cc. Inf. 1L. 5% dex. in D/W.
7 to 8	3000		1400	9	4025	20.25	10.358		.941	11.299	+8.951	+38.242	7.17	70.91		Ditto—with 1000 cc. saline and H_2O to make 3000 cc. Inf. 1L. 5% dex. in D/W.
Total						124.3	68.412	10.994	7.691	86.303						
Average		1121				17.757	9.773	2.199	1.099	12.329						
Apr. 19																Discharged.

TABLE VI

CASE F. R., MALE, AGE 55

Date	Fluid Output: Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N. Status Gm.	P.P. Gm. % A/G	Hem.	Wt. Kg.	Remarks
June 5													6.76	46.5	48.2	
8 to 9	3250	2100	1700	11.25	1270	2.8	20.403	7.355	.254	28.012	−25.212	−25.212				Tr. 500 cc. W.B. with reaction. Inf. 2L. 10% dex. in D/W; 1L. ditto, in saline.
9 to 10	4000	1810	1600	27.0	1370	6.0	11.021	3.297	.254	14.572	− 8.572	−33.784				Inf. 1L. 10% dex. in D/W; 2L. ditto. in saline; 1L. 5% amigen.
10 to 11	4500	2010	840	22.5	4090	22.385	8.767	9.856	.254	18.877	+ 3.508	−30.276	6.12	38	46.9	2L. 10% dex. in saline; 700. Gm. nutramigen in 500 cc. saline; H_2O to make total 2500 cc.
11 to 12	4500	2000	1300	22.5	4320	27.031	7.5	3.736	.254	11.490	+15.541	−14.735				Inf. 1L. 10% dex. in D/W; 700 Gm. nutramigen in 880 cc. pep. milk; water to make 4500 cc.
12 to 13	4000	1200	1000	16.92	4320	27.031	8.172	2.382	.254	10.808	+16.223	+ 1.488				Inf. 1L. 10% dex. in D/W; 700 Gm. nutramigen in 880 cc. pep. milk; H_2O to make 4L.
13 to 14	3500	—	1250	7.92	4155	28.385	8.64	—	.887	9.527	+18.858	+20.346				750 Gm. nutramigen in 880 cc. pep. milk; water to make $3\frac{1}{2}$L.
14 to 15	3000	—	1050	7.92	4150	28.385	7.835	—	.887	8.722	+19.663	+40.009	6.58	44	48.73	Ditto—water to make 3L.
15 to 16	3000		980	7.92	4150	28.385	7.64	—	.887	8.527	+19.858	+59.867				
16 to 17	3000		1100	7.92	4150	28.385	9.751	—	.895	10.646	+17.739	+77.606				
17 to 18	3000		1250	7.92	4150	28.385	8.642	—	.895	9.537	+18.848	+96.454				
18 to 19	3000		1040	7.92	4150	28.385	8.96	—	.895	9.855	+18.530	+114.984	6.84	46	52.15	
Total		9120				255.557	107.331	26.626	6.616	140.573						
Average		1824				23.222	9.766	5.325	.601	12.779						
June 25																Discharged.

TABLE VII

CASE V. B., MALE. AGE 39

Date	Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N. Status Gm.	P.P. Gm. % A	P.P. Gm. % G	P.P. Hem.	Wt. Kg.	Remarks
Nov. 10													3.95	2.6	45	64.54	
11 to 12	4500	950	900	13.5	3055				.7194								Inf. 1L. 10% amigen; 3L. 5% dex. in D/W; by tube 450 Gm. nutramigen in 500 cc. saline.
12 to 13	3500	1250	1180	18	3630	85.951	37.684	11.668	.7194	70.934	+15.017	+15.017					By tube 700 Gm. nutramigen —100 Gm. amigen in 2L. saline. Water to vol.
13 to 14	3500	1150	1200	18	3630				.7194				6.52		42		Ditto.
14 to 15	3000	650	860	9	3630				.7194								By tube 700 Gm. nutramigen; 100 Gm. amigen; 1000 cc. saline. Water to make 3L.
15 to 16	3000	450	800	9	3630	92.7	34.205	8.147	.7194	44.509	+48.191	+63.208					Ditto.
16 to 17	3000	400	860	9	3630				.7194								Ditto.
17 to 18	3000	—	940	9	3630				.7194				6.71		43	65.45	Ditto.
18 to 19	3000	—	800	9	3360	76.8	32.052		.7194	34.052	+42.748	+105.956					800 nutramigen in 1L. saline. Water to make 3000 cc.
19 to 20	3000	—	1400	9	4230				.7194								900 Gm. nutramigen in 1L. saline. Water to make 3L.
20 to 21	3000	—	1000	9	4700				.7194								1 Kg. nutramigen in 1L. saline and H_2O to make 3L.
21 to 22	3000	—	1000	9	4700	81	56.002		.7194	58.159	+24.998	+130.954					Ditto.
22 to 23	3000	—	1700	9	4700				.7194				6.93		44	69.25	Ditto.
Total		4850				336.451	159.943	19.815	8.633	207.654							
Average		808				28.037	13.329	3.303		17.304							

TABLE VIII

CASE P. F., MALE, AGE 41

Date	Fluid Intake Cc.	Fluid Output: Gastric Suction Cc.	Fluid Output: Urinary Vol. Cc.	Cl Intake Gm.	Caloric Intake	N Intake Gm.	—N—Output: Urinary N Gm.	—N—Output: Gastric N Gm.	—N—Output: Fecal N Gm.	—N—Output: Total N Output Gm.	N Balance Gm.	Cum. N. Status Gm.	P.P. Gm. % A/G	Hem.	Wt. Kg.	Remarks
Dec. 2													4.47/2.7	50	51.47	
3 to 4	4500	2400	15 5	31.5	3440	71.2	52.089	10.308	1.26	66.177	+ 5.023	+ 5.023				500 cc. W.B. (severe reaction) + 1L. 5% amigen; 1L. 10% dex. in saline + 600 Gm; nutramigen + 100 Gm. amigen in 1.5L. saline. Water to make 2.5L.
4 to 5	4500	2270	1620	27	3330				1.26				6.8	43		600 Gm. nutramigen; 150 Gm amigen in 3L. saline; H_2O to make 4500 cc.
5 to 6	4500	2140	1060	27	3330				1.26							Ditto.
6 to 7	4500	2070	950	27	3330	88.65	65.059	10.462	1.26	79.301	+ 9.349	+14.372			51.81	Ditto.
7 to 8	4500	2370	970	27	4035				1.26							750 Gm. nutramigen; 150 Gm. amigen + 3L. saline + H_2O to make 4500 cc.
8 to 9	4500	2450	1525	27	4035			5.154	1.26		+45.213	+59.585				Ditto; water to make 3500 cc.
9 to 10	3500	0	1120	9	4035	114.75	60.603		1.26	69.537						Ditto; amigen + nutramigen in 1L. saline; H_2O to make 3500 cc.
10 to 11	3500	0	1650	9	4035				1.26				6.7	41	52.73	Ditto.
11 to 12	3500	0	1050	9	4035				1.26							Ditto.
12 to 13	3500	0	1500	9	4035	153	83.99		1.26	89.030	+63.970	+123.555				Ditto.
13 to 14	3500	0	1650	9	4035				1.26							Ditto.
14 to 15	3500	0	1250	9	4035				1.26				6.81	42	55.68	Ditto.
Total						427.60	61.741	25.924	15.12	304.045						
Average						35.633	21.811	4.154		25.337						
Dec. 16																Discharged.

fusions, assuming that the total protein concentration was uniformly 7 Gm. per cent. Five hundred cubic centimeters of whole blood would thus yield about 250 cc. of plasma, which would contain 17.5 Gm. of proteins, equivalent to 70 calories and 2.8 Gm. of nitrogen. The protein contained in the hemoglobin of the transfused red cells was not included.

Like the daily fluid intakes, the daily caloric intakes in the control group fluctuated widely, from 80 calories for the 3rd and 4th days of F. W. (Table III) to 2,200 calories for the 3rd and 5th days of R. B. (Table II). In fact, R. B. had almost the basic caloric requirements throughout his convalescence, the only one who approached this caloric intake in the control group. The nitrogen intakes varied still more widely, ranging from 0 for three days for F. W. (Table III), for four days for J. S. (Table IV), and for four days for D. R. (Table I, to 11.11 Gm. for the 6th day for R. B. (Table II), the averages being 3.31, 6.12, 5.5 and 3.97 Gm. for these four cases, respectively.

In the feeding group, except for the first day in the case of A. V. (Table V), and the first and second days in the case of F. R. (Table VI), in all the other postoperative days the intake was on the luxury level. The respect for the tradition of withholding feedings for the first few days postoperatively in abdominal cases, accounted for the "lean" first day for A. V., and first two days for F. R. In our last two cases, V. B. (Table VII), and P. F. (Table VIII), feeding was started after the first 12 hours, which period may still be shortened, with courage gained from further experience. Except for the three "lean" days mentioned above, the caloric intake in this group ranged from 3,050 to 4,700 (V. B.) daily. It may be mentioned that this latter amount was given in response to complaints of hunger on the part of a patient, who was being fed 3,360 calories a day—a phenomenon connected with convalescence which bears further investigation.

The nitrogen intake in the feeding group, except for the three "lean" days mentioned, ranged from 20.25 Gm. (A. V.) to 38.25 Gm. daily for the 5th, 6th and 7th days of P. F. The average daily intakes were 17.7, 23.22, 28.05 and 21.81 Gm., respectively, for the four feeding cases.

In the feeding group, the caloric and nitrogen values were mostly derived from amigen and nutramigen* in the feedings, although dextrose and amigen injections and blood transfusions contributed some part of the first days' intake. Amigen is a casein enzymatic digestate containing approximately 85 per cent amino-acids and 15 per cent polypeptides, each gram yielding 3.4 calories and 0.12 Gm. of nitrogen. Nutramigen contains, in addition to amigen, dextri-maltose, a neutral fat, arrowroot starch, calcium gluconate and brewer's yeast, and minerals added to simulate the quantities present in cow's milk. Nutramigen has been developed as a food for infants and is marketed as such. It yields 4.7 calories per gram and contains 2.7 per cent nitrogen. In preparing the feeding, the amigen and nutramigen were mixed

* Both amigen and nutramigen were kindly supplied us by the Mead-Johnson Co.

with a convenient amount of physiologic saline or water to make the mixture easily instillable by means of a syringe through the feeding tube into the jejunum. The feedings were so spaced that from 50 to 150 cc. was given every hour. A larger amount might cause nausea and vomiting, either as a result of distention or of too rapid an absorption resulting in hyperaminoacidemia. The balance of the mixture was kept in the refrigerator, since it is a fertile medium for bacterial growth, and decomposition easily sets in.

Collection and Care of Specimens.—The 24-hour urine was preserved with thymol, measured, and kept on ice. In some cases in both groups, both urine and gastric suction specimens were sometimes pooled for two or three days and an aliquot part of the pooled specimens taken for nitrogen determination. In these instances the total nitrogen over the number of pooled days was recorded in the tables as one figure. The Wangensteen drainage (gastric suction) was preserved in sulfuric acid. Likewise preserved in sulfuric acid, were the stools collected and pooled over the study period. The discharges from the wounds were never large enough to be taken into consideration, random samples yielding negligible amounts of nitrogen.

The samples of blood for blood plasma protein determinations were taken in one of two ways. Where the specific gravity method of Barbour and Hamilton[11] was used, the anticoagulant used was heparin. Where a chemical method was used, the anticoagulant was sodium oxalate. The hematocrit was determined in the Sanford-Magath tube, and in most cases was read each time the plasma proteins were determined.

Chemical Methods.—As stated above, when the total plasma protein concentration only was needed, it was determined by the specific gravity method of Barbour-Hamilton,[11] using the apparatus designed by these authors. Where it was desired to obtain the albumin and globulin figures, the method of Wu and Ling as modified by Greenberg[12] was used.

In determining the nitrogen of the urine, stool and gastric drainage, the method of Rappoport as modified by Levy and Palmer[13] was used.

ANALYSIS OF THE TABLES

The fluid, caloric and nitrogen and NaCl intakes have been discussed in a foregoing section of this paper.

Fluid Output. (Gastric Suction)—By this item is meant the amount of drainage, in cubic centimeters, yielded by the Wangensteen apparatus each 24 hours. The suction was kept up for from four to seven days. The amount in cubic centimeters varied from a minimum of 400 cc. (V. B.) to a maximum of 2,800 cc. (A. V.). As mentioned before, this source of fluid loss complicated the problem both of fluid and of sodium chloride replacement. The drainage of J. S. contained visible blood for the second and third days. The amount of nitrogen lost from this source will be discussed under the heading of *"Gastric Nitrogen."* As a rule, the suction tube was left

in place for a shorter time in the control cases than in the feeding cases, in whom it was needed for feeding.

Urinary Output.—As might be expected from what has been said of the fluid intake, there was more fluctuation of the urinary volume in the control group than in the feeding group. Generally speaking, however, the urinary volume was quite adequate. The smallest output was 650 cc. for the first day of A. V., the first feeding case, when no provision was made for the gastric drainage. The fluctuations in the control group existed throughout the course of the convalescence, while in the feeding group the urinary volume was fairly well stabilized to from 1,000 to 1,900 cc. after the gastric suction was discontinued. The widest fluctuations were in the case of F. W., a control case, whose output ranged from a low of 700 cc. to a high of 3,550 cc. Incidentally, he also had a low blood plasma protein concentration of 5.23 Gm.% at the time of this excessively high output.

Urinary Nitrogen.—In the control group, except for R. B., the nitrogen excretion in the urine averaged respectively 13.69, 15.46, 18.03 and 11.74 Gm. daily. These figures are in the neighborhood of urinary nitrogen figures given for the ten days of complete starvation, as found in Succi, Cetti and Levanzin.[14] They are, except for R. B., all above the 13 Gm. given as the nitrogen excretion of average persons. Brunschwig, *et al.*, gave figures for two cases of gastric resection, one, excreting 73.53 and the other 175.79 Gm. of nitrogen in ten days, averaging 7.35 and 17.58 Gm. respectively. Just how much of this excreted amount is "toxic loss," how much is starvation loss, as modified by previous undernutrition, is not clear.

In the feeding group, A. V. was taking an average of 17.78 Gm. of nitrogen and excreting an average of 11.21 Gm. daily; F. R. was taking in 23.22 and excreting 9.77 Gm. daily; V. B. was ingesting 28.04 and excreting 13.33 Gm., while P. F. was ingesting 35.63 and excreting 21.81 Gm. These figures suggest that A. V. and F. R. were ingesting perhaps less than they could fully utilize; that V. B. was perhaps getting the optimum amount, and that P. F. was perhaps ingesting an amount above the optimum. Both F. R. and V. B. had blood transfusion reactions, which no doubt accounted for the large amount of urinary nitrogen excreted during the days immediately following the transfusion. This would tend to introduce an error in the amount of nitrogen actually lost from the body.

Gastric Nitrogen.—This item represents the amount of nitrogen lost in the Wangensteen drainage. In the control group, the figures were, minimum .771 Gm. (D. R.), maximum 25.368 Gm. (J. S.), for any one day. The blood in the drainage of J. S. accounts for a part of this nitrogen. The average daily loss was 3.18, .815, .216 and 17.45 Gm. Aside from hemorrhage as a source of nitrogen loss, it is quite possible that the oozing of exudate from the operated viscus may also have contributed to this loss. In the feeding group, the average daily losses were higher, being 2.2, 5.33, 3.3 and 4.15 Gm., respectively, but how much, if any, of it represented refluxed feeding from the jejunum has not been ascertained.

Even taking the loss in the control group as the more nearly actual figure, it is clear that a considerable amount of nitrogen can be lost from this source.

Fecal Nitrogen.—The fecal nitrogen in both of these groups is low, with a tendency in the feeding group to be slightly higher. The only subject who almost approximated the classical average daily norm of 1.3 Gm. was V. B., in the feeding group, with an average of 1.25 Gm. of nitrogen excreted in the stools. In the control group, the lowest daily average was .21 Gm. (J. S.), and the highest 0.815 Gm. In the feeding group, the lowest daily average was 0.601 (A. V.) and the highest 1.26. These low figures are suggestive of the low fecal nitrogen excretion of Benedict's[15] subjects under prolonged restricted feeding.

Total Nitrogen Output.—The daily total nitrogen output averaged 13.69, 16.4, 9.24 and 20.13 Gm. daily in the control group, and 12.33, 12.78, 17.3 and 15.38 Gm. in the feeding group. In the control group, therefore, the average total nitrogen loss of two cases is significantly higher than that found in complete starvation. The day-to-day output, however, fluctuated a great deal, and in three out of four control cases there were always some days when the output was considerably higher than that found in starvation.

The Nitrogen Balance and the Cumulative Nitrogen Status.—Except for the first day in the case of F. W., none of the four cases in the control group achieved nitrogen balance during any of the days under study; the nearest approximation to a balance was a loss of 0.61 Gm. of nitrogen on the part of F. W. on the 12th postoperative day. The average daily nitrogen losses were 10.37, 10.28, 3.73 and 16.4 Gm. daily. The sum-total of these losses were 114.09, 82.21, 44.8 and 141.11 Gm., respectively. This cumulative deficit is entered under the column entitled "Cumulative Nitrogen Status" in the tables.

In the feeding group, except for the three days of withholding feeding, positive balances were registered on all the days. The cumulative nitrogen surplus, after subtracting the deficit of the three "lean" days, were 38.25, 114.88, 130.95 and 123.56 Gm., respectively. This surplus is also entered under the heading of "Cumulative Nitrogen Status."

The Hematocrit and Plasma Protein Determinations.—In the absence of blood volume determinations and in the presence of multiple infusions, especially in the control group, these determinations have been robbed of much of their absolute value. Generally speaking, however, the tendency of the plasma protein level was downward in the control group, and upward or relatively stationary in the feeding group. This finding in the feeding group is more significant than the figures in the control group because of the better regulated fluid intake and the comparative absence of infusions. The sharp upswing of the total plasma proteins and hematocrit from 5.35 to 7.04 Gm.% in the case of F. W. is a bizarre phenomenon. Whether it is due to an hemoconcentration or to a rise of blood proteins, even while the body was still losing nitrogen, is only a matter of speculation.

In the case of the hematocrit values, the tendency of both groups is to

dip below the preoperative figures, showing that in spite of the transfusions, there is some loss of blood. This is particularly true with those cases in which there have been blood transfusions reactions (F. R. and V. B.).

The Weight Curve and the Cumulative Nitrogen Status.—If a table is constructed by posting the cumulative nitrogen status during the period in one column, the equivalent of this in body tissue in a second column, and the gain or loss of body weight in a third column, a better picture of the relationship of these three factors may be seen. The equivalent of the nitrogen gain or loss in body tissues is arrived at by multiplying the nitrogen deficit or surplus by the standard factor of 6.25, and multiplying the result, in turn, by five, assuming that the protein is deposited with 80 per cent water to form body tissues. Table IX represents this relationship in the eight cases studied.

TABLE IX

Patients	Cumulative Nitrogen Status Gm.	Body Tissue Lost or Gained Gm.	Body Weight Lost or Gained Gm.
D. R.	−114.09	−3455	−6950
R. B.	− 82.21	−2569	−4300
F. W.	− 44.8	−1397	−3430
J. S.	−141.13	−4410	−5230
A. V.	+ 38.24	+1195	+1540
F. R.	+114.98	+3593	+3950
V. B.	+130.95	+4092	+4710
P. F.	+123.56	+3861	+4210

It will be seen from the table that while no linear relationship exists between the nitrogen deficit or surplus and body weight loss or gain, yet the trends are roughly parallel. Figures 1 and 2 represent this relationship graphically. Both the charts and the tables show that in the control group there was a significant loss of body weight, while in the feeding group there was a significant gain. Another interesting point in connection with body weight in the control group is that it required a fairly long time for the initial weight to be restored. Thus D. R. on discharge, 35 days postoperatively, was still 900 Gm. under the initial weight; R. B. almost reached his initial weight three weeks postoperatively; F. W. two weeks postoperatively was still 4,450 Gm. under his initial weight; and J. S. on the 17th postoperative days was 3,870 Gm. below her initial weight.

Of the two cases in the control group who were followed for 12 days, D. R. lost 115 Gm. of nitrogen and 6,950 Gm. of body weight, and F. W. lost only 45 Gm. of nitrogen and 3,430 Gm. of body weight. It is interesting to observe that two fasters—Succi (Florence, 1888), in 12 days of complete fasting lost 134 Gm. of nitrogen and some 7,700 Gm. of body weight, while Levanzin lost, in the same period, 120.53 Gm. of nitrogen and 6,760 Gm. of body weight. The weight losses of three other fasters for 12 days were—Jacques (1888) 6,970 Gm., Beatue (1907) 6,410 Gm. and Schenk[14] (1906) 6,800 Gm. It is apparent that in spite of the infusions and transfusions given to the patients undergoing gastric resection, the nitrogen and body weight losses of some cases can approximate those of complete fasting.

Theoretically, the body weights of patients with hypoproteinemia may be higher than the actual weight, as a result of the loss in colloid osmotic pressure of blood proteins and the retention of water by the tissues (latent edema). In such a circumstance, the body weight would be sustained at a falsely high level, to undergo a sudden drop with restoration of colloid osmotic pressure to the blood. With D. R. there was no suggestion of this phenomenon taking place. With F. W., the only other patient with definite hypoproteinemia, the loss of only 260 Gm. of body weight in the first three days, in the face of a blood protein which fell from 6.3 to below 5.5 Gm. per cent, and the loss of 2,130 Gm. on the fourth day after the excretion of 3,550 cc. of urine on the previous day, is suggestive of this mechanism having been operative.

The Period of Hospitalization.—This study was not carried out with any planning, but was really an afterthought. A number of factors entered into determining when this period should be terminated, among them whether a patient has relatives at home to take care of him after his discharge. It is apparent that a patient living alone must stay in the hospital until he is stronger. In a future series, it is planned to have a better control of this factor. Meanwhile, the number of days of hospitalization of these two groups are interesting:

Control Group	Days
Name	
D. R.	35
R. B.	21
F. W.	21
J. S.	17
Av.	23.5
Feeding Group	
A. V.	22
F. R.	17
V. B.	14
P. F.	14
Av.	16.75 days

There is, thus, a shorter hospitalization period of 6.25 days in the feeding than in the control group.

Infusions and Transfusions: The number of infusions and transfusions administered to the patients in these two groups are shown in Table X.

TABLE X

Control	No. of Days of Infusion	Total Infusions Given—L.	No. of Transfusions	Total Amount of Transfusions Blood Cc.	Plasma Cc.
D. R.	10	18	4	1,000 cc. W.B.*	1,000 cc.
R. B.	7	19.5	6	1,500 cc.	1,050 cc.
F. W.	2	7	1	500 cc.	
J. S.	5	12	1	500 cc.	
Feeding					
A. V.	2	3	1	500 cc.	
F. R.	4	12	1	500 cc.	
V. B.	1	4	0	0	
P. F.	1	2	1	500 cc.	

*W. B. = Whole blood.

It will be seen that both the number of infusions and transfusions were markedly reduced in the feeding group. This was also the experience of Stengel and Ravdin,[16] with their orojejunal feeding cases. In the last two patients, V. B. and P. F., the infusions were given in the first 12 postoperative hours, and V. B. had no transfusions, while the transfusion in P. F. as a result of the severe reaction seemed to have been more harmful than otherwise, reducing the hematocrit value significantly, and could well have been dispensed with. The greater comfort to the patient in the use of fewer infusions and the economic advantage of reducing the number of transfusions are considerable.

Postoperative Asthenia.—While the usual postoperative debility was present in the control group, it was interesting to note that it was minimal in the feeding group. This was most apparent in the last two feeding cases, V. B. and P. F., who got restless and asked to get up in a wheel chair on the eighth day. On the twelfth day they were helping move beds and weighing fellow patients. This absence of postoperative debility was also observed by Brunschwig, *et al.*, in one case, a patient who had gained 20.39 Gm. of nitrogen. Although this absence of asthenia seems to be definite in this series of four patients, it is still impressionistic. In a future series we propose to study this subject objectively.

Disadvantages of the Feeding Method.—The main disadvantage of this method of feeding is the discomfort caused by the presence of the nasal tube. This discomfort is relieved in 24 hours after the tube is removed. The other disadvantage is the objectionable taste of the mixture. When the mixture is administered through a tube, this difficulty is circumvented, but many patients rebel against taking it orally. If the patient is told that it is a "build-up medicine" which he will not have to take for more than a week (after the five to six days of tube feeding), however, he can usually be persuaded to "down" the mixture. Occasionally diarrhea occurs. Whether this is due to overfeeding or to other factors has not been determined.

In this series of four feeding cases, and in a large series of feeding with amigen and nutramigen to patients who were not under study, there was never any distention encountered.

DISCUSSION AND COMMENTS

The question arises, how does this attempt to maintain caloric and nitrogen equilibrium postoperatively differ from previous attempts? None of the technics employed in this study are essentially new. Various technics of postoperative tube feeding have been periodically reported. Elman and Brunschwig showed the feasibility of achieving nitrogen balance by parenteral feeding, and this has been repeated by Landesman and Weinstein.[17]

Stengel and Ravdin,[16] in 1939, and Ravdin and Stengel,[18] in 1940, reported an orojejunal method of feeding, using the Abbott tube, and an ingenious automatic feeding machine. Unfortunately, there was at that time no ready-made homogenous mixture available. Furthermore, no nitrogen balance

was done, and without knowing the gravity of nitrogen losses, no adequate replacement could be given, particularly as these authors emphasized only the necessity of a basal intake.

The first point of departure in this work from previous work is the utililization of the absorbing surface of the reaches of the intestines beyond the point of operative trauma for the absorption into the system of a feeding mixture which requires the minimum of digestive effort. The combination of the feeding tube and this mixture obviates the difficulties which bar the way to immediate posoperative feeding, especially in abdominal cases, namely, the vomiting, anorexia and impairment of the digestive processes caused by an operation. The second point of departure is the strict adherence to a caloric and nitrogen intake, which would at least fully replace the losses resulting from the operation.

To rely solely upon intravenous alimentation to do this is to put to heroic use a method which, at best, must remain an excellent adjuvant. Elman and Brunschwig both realize this, Elman saying: "The purpose of parenteral alimentation, in surgery at least, is to clear temporary hurdles"; and Brunschwig, *et al.*, stated: "Minimal caloric requirements, including sufficient protein (as amino-acids), may be met by intravenous nutrition." Thus, in order to give 1,749 calories, and a nitrogen intake of 18 Gm., Brunschwig gave as much as 4,500 cc. of fluid intravenously in six hours, an amount which must tax the circulatory system by the production of an acute plethora. And, unfortunately, the caloric and nitrogen requirements postoperatively are not often basal, as has been demonstrated by Cuthbertson,[6] An analysis of the four cases reported by Brunschwig, *et al.*, as having registered a net gain of nitrogen ten days postoperatively, shows that only one achieved a positive nitrogen balance as a result of intravenous feeding. In Elman's report of 35 patients, in whom nitrogen balance was achieved by intravenous alimentation, only four postoperative cases were reported in detail, and in none of these four was a positive nitrogen balance consistently achieved. Recently, Gardner and Trent[19] found similar difficulty in maintaining nitrogen balance by intravenous alimentation in patients with large nitrogen losses postoperatively. If to this limitation may be added the dangers of pyrogenic reactions, and of phlebitis and thrombosis of veins, the restoration of whose patency is never certain and which could be conserved for use in greater emergencies, the present tendency to rely mainly on intravenous infusions to supply caloric and nitrogen requirements seems unjustifiable.

Finally, two further points must be emphasized. The first is that blood and plasma transfusions, dramatic as they are in the restoration of the blood volume in acute hypovolemia caused by hemorrhage or acute loss of plasma proteins, cannot be depended upon as the sole caloric and nitrogen supply for the body. Sachar, Horvitz and Elman[20] are of the same opinion, and a little arithmetical calculation will show the strength of this statement. As shown above, the amount of plasma proteins in 500 cc. of transfused blood will yield

70 calories and 2.8 Gm. of nitrogen. To supply the basic caloric requirement alone would require 26 transfusions a day! Were all the hemoglobin in the transfused blood also utilized for body energy, for which we have no evidence at present, only 55 Gm. of protein in the form of hemoglobin would

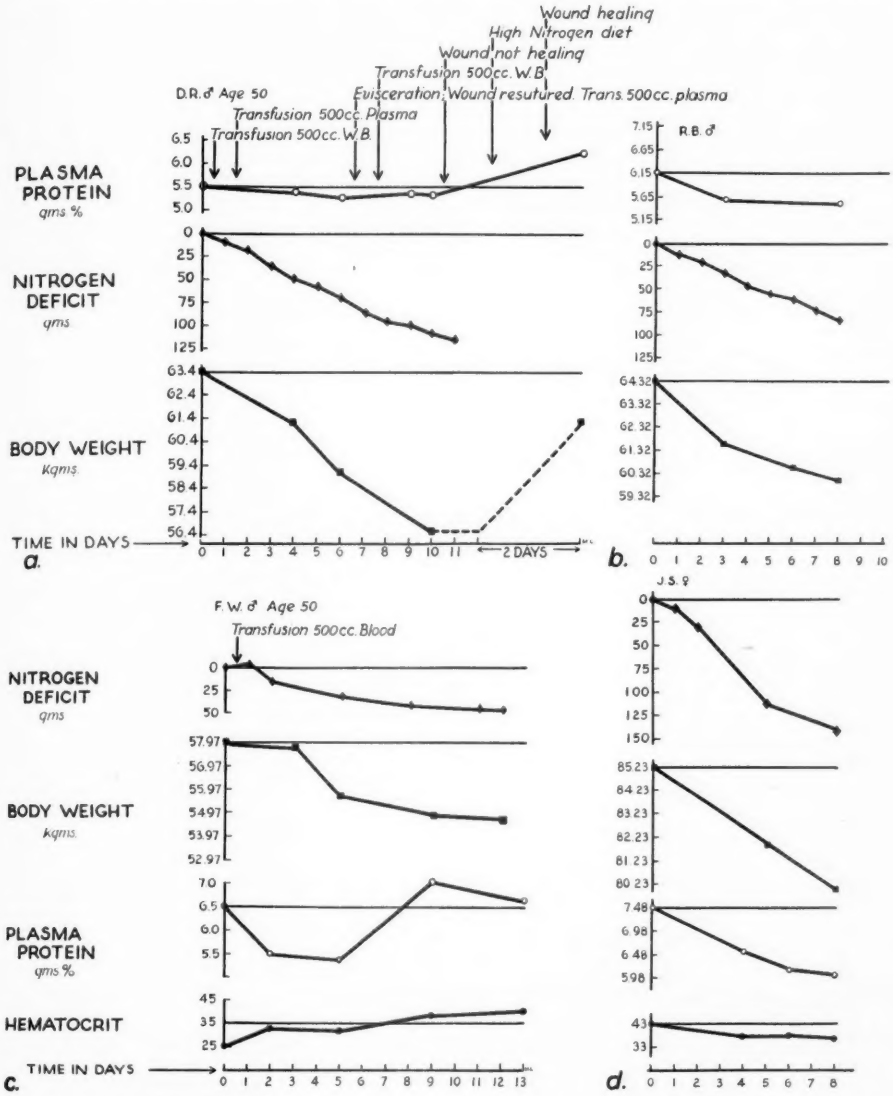

FIG. 1.—Panels a, b, c and d are graphic records of the plasma protein, nitrogen deficit, body weight and hematocrit curves (except two) of members of the control group; namely, of D. ., R.B., F.W., J.S., respectively. Note particularly the downward trends in both the nitrogen curve and the body weight curve.

be available, making a total of 72.5 Gm., and to satisfy the basal caloric needs under these hypothetic circumstances would require six transfusions a day.

The other point, which Case D. R. suggests, is that unless provisions are made for full caloric and nitrogen maintenance postoperatively, patients

coming to operation with plasma protein concentrations near the "lower limit of normal" may be poor operative risks, for this so-called lower limit of normal already reflects some depletion of body proteins, which would be aggravated by the "toxic loss" of nitrogen and starvation incident to an

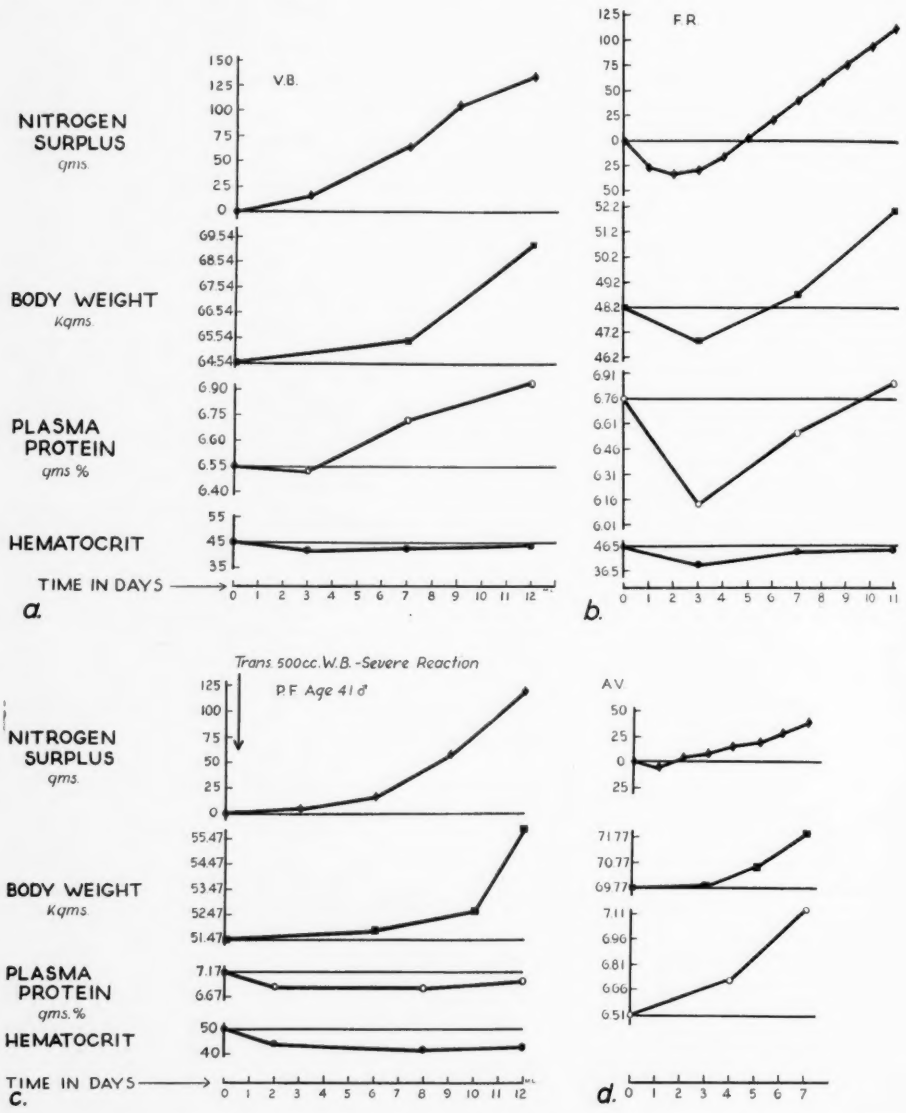

FIG. 2.—Panels a, b, c and d are graphic records of the nitrogen deficit, body weight, plasma protein and hematocrit (except one) curves of members of the feeding group, namely, of V.B., F.R., P.F., and A.V., respectively. Note the consistent upward trend in nitrogen surplus and body weight in all the four cases. The plasma protein curves are consistently upward in two and show a slight depression in two.

operation, leading to impaired wound healing. It may be safer for surgeons to set a higher "lower limit of normal" to surgical patients than is required in other specialties.

It must be emphasized that these four cases are only preliminary, and point the way to a wider application of at least full caloric and nitrogen replacement in surgical patients. A number of refinements have still to be worked out, among them suitable feeding technics for different types of cases, and the optimum postoperative intake, and objective tests for strength and endurance.

SUMMARY AND CONCLUSIONS

(1) The nitrogen balance, the body weight and the plasma proteins of four patients with duodenal ulcer, and undergoing partial gastrectomy, and treated postoperatively by the standard ward routine, were followed for from 7 to 12 days. There was found to be in all these cases a cumulative nitrogen deficit, a progressive loss of body weight and a suggestive progressive fall of plasma protein concentration.

(2) In another group of four patients with similar pathology, and undergoing the same operation, in whom the caloric and nitrogen balance was maintained by tube feeding with an easily assimilable feeding mixture, there were achieved a positive nitrogen balance, a cumulative nitrogen surplus, and a progressive rise of body weight and of plasma proteins. There also seemed to be the minimum amount of postoperative asthenia.

(3) The technics employed in this study were discussed, as well as the fallacy of expecting to maintain nutrition by blood transfusions, and the necessity of a higher "lower limit of normal" of plasma proteins for surgical patients.

(4) The preliminary character of this work is emphasized.

The authors wish to acknowledge their grateful thanks to Dr. Victor Carabba who performed a number of these gastrectomies.

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PREOPERATIVE SCRUBBING IN ABDOMINAL SURGERY

I—EXPERIMENTAL STUDIES*

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ABOUT 35 YEARS AGO Theodore Kocher[1] emphasized the value of preoperative scrubbing in the preparation of a patient for surgical operation. In his text book of Operative Surgery he makes the following statements: "It is remarkable how many people of the present day refuse to be convinced that dirt can be really and readily removed by means of simple running water. It is no uncommon sight to see the surgeon and his assistants scrupulously careful about the cleansing and scrubbing of their own hands, while the patient's skin is washed with a little soap and lotion immediately before the operation, receiving subsequently a few doses of corrosive lotion, proceedings which imply a contradiction.

"There is no reason why every patient should not have the benefit of a vigorous cleansing (soap and water) from head to foot in the surgical sense of the term. This washing, which is carried out in a warm bathroom the day before operation, should be preferred to the practice of some surgeons who apply antiseptic poultices to the skin at the site of the operation." Many surgeons still follow Kocher's advice.

It is difficult to prove clinically whether or not preoperative scrubbing is effective. However, in the experimental animal this can be ascertained more accurately. At the outset, we should define our terms, and by scrubbing we mean the use of a small, soft hand brush with soap and unsterilized warm water applied to the part vigorously for about eight minutes. The term 'preoperatively' is used to include a period up to 12 hours before operation. Immediate surgical sterilization is not included in this study.

In order to determine the efficacy of scrubbing, we decided to test the method on guinea-pigs and dogs. At the same time, a group of patients were treated by scrubbing, in an effort to evaluate the method clinically. These results are to be reported in a supplementary paper.

EXPERIMENTAL STUDIES

First, we wanted to establish normal standards so that variations could be charted. Daily leukocyte counts and rectal temperatures were recorded in 15 guinea-pigs. Our conclusions were that leukocyte counts and body temperatures are not stable in guinea-pigs, varying with room temperature

* Aided by a grant from the Eli Lilly Research Fund.

and other factors not determined. These reactions are also not reliable guides in evaluating the general condition of an animal with normally healing or with infected wounds. There is, however, one significant finding in scrubbed guinea-pigs which is only rarely observed in the controls. This is an increase in the number of leukocytes, averaging about 7000 per cu. mm. immediately after operation (Chart 1). Although some animals die of

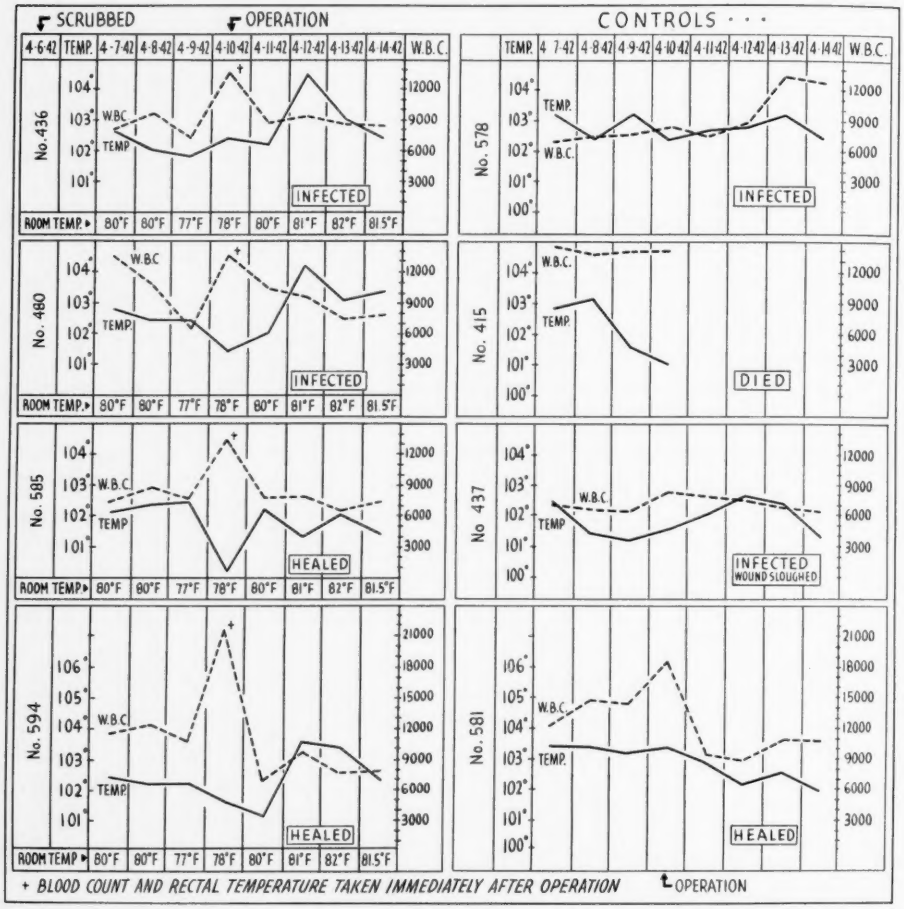

CHART 1.—Charts showing room temperature, body temperature and leukocyte counts in four guinea-pigs which had been scrubbed 96 hours prior to operation and four controls. Three-tenths of a cubic centimeter of a pure culture of *Staphylococcus aureus* was introduced into the abdominal wound at the time of operation. Note the increase in leukocytes immediately after operation in the scrubbed pigs.

staphylococcic septicemia after inoculation, showing leukocyte counts of 500 or less, there was usually a leukocytosis, and the body temperatures remained high. In environmental temperatures of 70° to 80° F. infected guinea-pigs were found to have temperature, ranging up to 106° F.

NORMAL RATE OF HEALING

The abdominal wall of guinea-pigs was shaved, cleaned with soap and water, and antisepticized with ether and tincture of methiolate. Aseptic

technic was used, and operations were performed under ether anesthesia. An incision five centimeters long was made in the abdominal wall, extending through the skin, subcutaneous tissue and abdominal muscles. The peritoneum was not opened. Incisions were closed with interrupted silk sutures in the muscle layers, subcutaneous tissue and skin. In the first group of pigs, bandages were applied and the legs fastened with rubber bands, as suggested by Du Mortier.[2] However, this was found to be impractical, as the pigs developed edema of the legs and back and if not restrained they tore the dressings off. We, therefore, decided to observe the normal healing without dressings. The pigs were accordingly returned to their clean cages immediately after operation. Fourteen pigs were studied. Eleven wounds healed *per primam*. Three of this group, or 21.4 per cent, developed infections in the wound. The word 'infection,' as used here, implies any evidence of bacterial action in the tissues, either grossly or microscopically. On gross examination, none of these wounds were thought to be infected for there were no clinical signs to indicate this. Microscopically, some of them did show evidence of infection.

This study, which was repeated three times, with similar results, revealed that abdominal wounds in guinea-pigs usually heal without infection when made under the aseptic technic described, even though dressings are not applied. Since some of the animals were operated upon during the summer and some in the winter, the effect of high and moderate temperatures on wounds could be observed. It was found that wounds heal in 4–6 days (average 5+ days) in moderate room temperature (70°–80° F.). In high external temperatures (100°–110° F.) wounds required 7–13 days (average 9+ days) for healing.[3] Low temperatures were not tried. This observation may be important in the present crises, for our forces are stationed in parts of the world where temperatures vary greatly. Since the body temperature of the pig is elevated proportionately to room temperature, we may deduce that high body temperatures retard, but do not prevent healing.

REACTION OF FRESH WOUNDS TO CONTAMINATION BY STAPHYLOCOCCUS AUREUS

Having determined the normal reactions of guinea-pigs to incisions made under aseptic technic, we next wished to learn the effect on the wound when a pure culture of *Staphylococcus aureus* was introduced. Such a culture was obtained from a patient with a carbuncle. The organism was grown six hours upon chopped meat medium, transferred every 24 hours, and occasionally plated on blood agar to check for purity. Virulence of the organism was determined by injecting the culture subcutaneously into guinea-pigs. The smallest dose which uniformly produced an abscess without causing death of the animal, when a six-hour culture was used, was found to be 0.3 cc. Abscesses were checked by microscopic examination. Virulence was maintained with surprising uniformity. Before every group of experiments the organism was injected into guinea-pigs and then reisolated from the resulting abscesses. Direct inoculation of the fresh wound was made by placing 0.3 cc.

of the six-hour culture of *Staphylococcus aureus* into the incised abdominal muscles. Incisions and closures were performed exactly as in the controls. Sixty-seven and one-half per cent of the animals developed infections in their wounds.

Many experiments were now undertaken in an effort to repeat the findings of Du Mortier,[2] who demonstrated the high degree of immunity which develops in granulation tissue. Also, studies were made to determine the extent of the local immunity by making multiple incisions at various sites in the abdominal wall at different times. By this method we were able to prove that the immunity in a wound does not extend beyond the area of granulation tissue. These experiments will be published subsequently.

THE EFFECT OF SCRUBBING UPON THE PRODUCTION OF WOUND IMMUNITY

The body reacts locally by inflammation and exudation whether the injury is produced by mechanical, bacterial, chemical, or any other trauma. We thought that perhaps scrubbing the abdomen might be a factor in the production of immunity. Thirty-four guinea-pigs were studied. They were anesthetized with ether, and their abdomens were shaved and then scrubbed with a hand brush such as is used by surgeons in the scrub room. This was done for five to eight minutes depending upon the reaction of the skin. Care was taken not to bruise or injure the abdominal wall. At the end of 24, 48, 72, 96, 120, and 144 hours after scrubbing, an incision was made in the abdominal wall under aseptic technic, exactly as previously described, and 0.3 cc. of a culture of *Staphylococcus aureus* was introduced. The experiment was then repeated, scrubbing the abdominal wall on six successive days. On the 6th day, all pigs were operated upon and the culture introduced as previously described. Four controls were operated upon without scrubbing in each series.

Seventy-five per cent of the guinea-pigs scrubbed 24, 48 and 72 hours before operation were infected. Fifty per cent of the guinea-pigs scrubbed 96 and 120 hours became infected. Sixty-seven and one-half per cent of the controls were infected.

In all instances the wound bled much more freely in the scrubbed pigs than in the controls. Grossly, the scrubbed pigs seemed to have infection in only 20 per cent. On the 6th postoperative day sections were taken from the abdominal wounds. Examination of these sections microscopically failed to reveal any consistent discernible difference in the degrees of infection in the scrubbed and unscrubbed pigs (Fig. 1). When the experiments were repeated the difference again was not in individual wounds but in the per cent of animals infected.

In order to check our findings, four guinea-pigs were prepared in the manner described above. Instead of using a six-hour culture we used a 24-hour culture of *Staphylococcus aureus*, and instead of 0.3 cc. we used 0.5 cc. in the incision. A second group of eight pigs was scrubbed 96 hours previous to operation, using the same culture. One of the controls died within 24

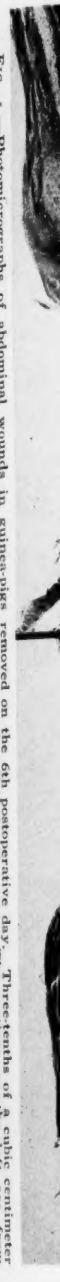

FIG. 1.—Photomicrographs of abdominal wounds in guinea-pigs removed on the 6th postoperative day. Three-tenths of a cubic centimeter of a pure culture of *Staphylococcus aureus* was introduced into the wound at the time of operation.

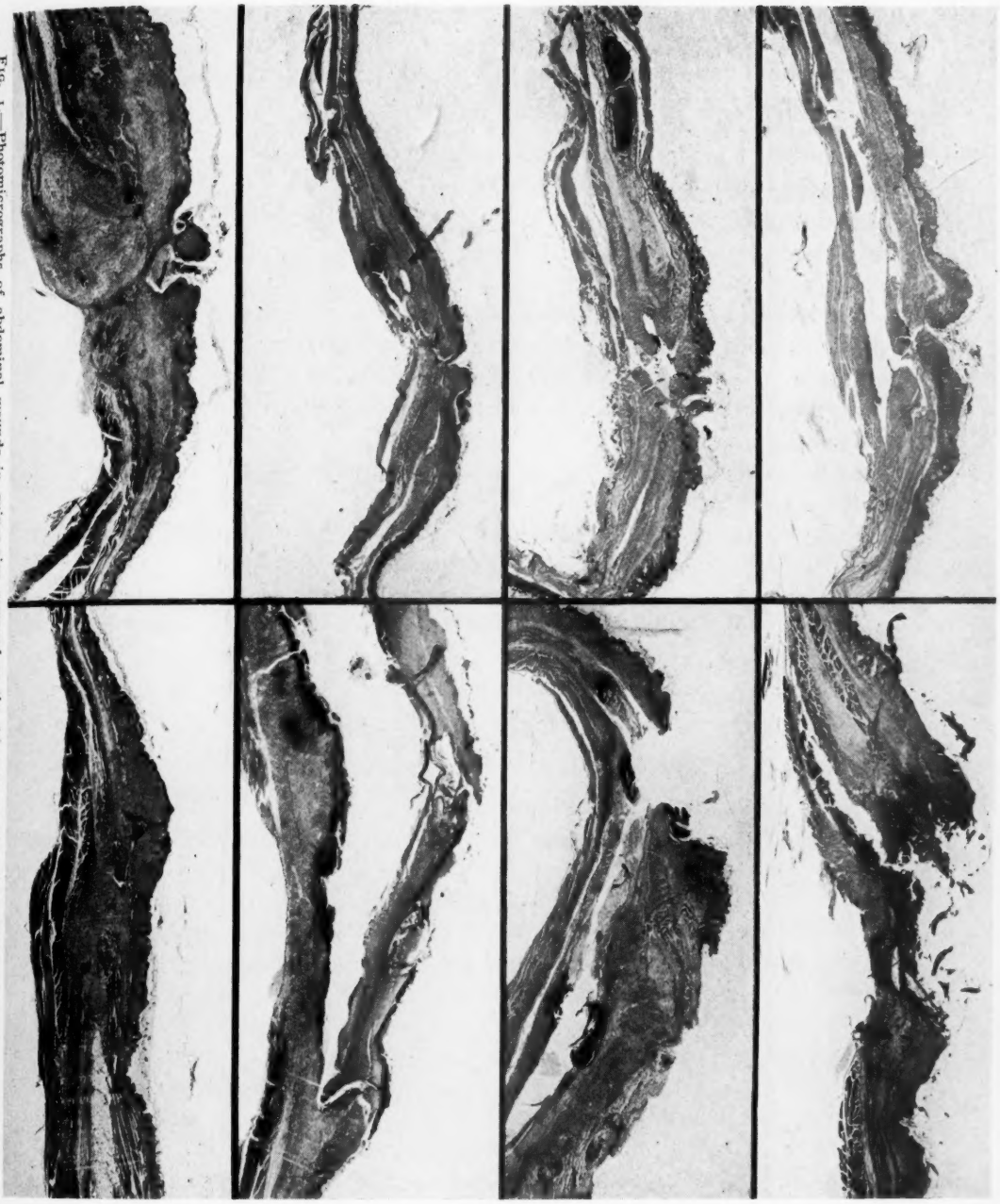

FIG. 1.—Photomicrographs of abdominal wounds in guinea-pigs removed on the 6th postoperative day. Three-tenths of a cubic centimeter of a 24-hour culture of *Staphylococcus aureus* was introduced into the wounds, as described in the text. The sections on the left are from

hours of a staphylococcus septicemia. Another died on the 4th postoperative day with a large abdominal abscess. The remaining controls survived but developed wound infections. All of the animals which had been scrubbed 96 hours previously showed infections in their wounds (Fig. 2). It is apparent from this study that the size of the dose plays an important rôle in the resulting infections.

EFFECT OF SCRUBBING THE ABDOMEN UPON PERITONEAL IMMUNITY

A group of eight guinea-pigs was now studied for intraperitoneal immunity. All operations were performed as previously described except that the peritoneum was opened. Three-tenths of a cubic centimeter of a pure culture of *Staphylococcus aureus* was introduced and the abdomen closed with interrupted silk sutures in layers. Five of the animals developed wound infections. All recovered. At autopsy, none showed any evidence of peritonitis.

Another group of eight pigs was studied in a similar manner except that foreign bodies, such as 18-inch knotted strands of sterile silk suture, were introduced in the peritoneal cavity. By this technic it was possible to produce a small abscess about the suture which was entirely localized by the omentum and adhesions when the animals were autopsied. Four of the abdominal wounds became infected although the culture was introduced into the abdomen with a glass pipette. One pig died of peritonitis and wound infection.

The same experiment was repeated with preoperative scrubbing. There was no difference in the intraperitoneal reaction from the control. However, only about 50 per cent of the pigs scrubbed 96 hours before operation developed wound infections.

SCRUBBING EXPERIMENTS UPON DOGS

In an effort to further study the problem we decided to perform similar experiments upon dogs. Eight healthy animals were anesthetized with ether, the abdomen was clipped, shaved and scrubbed for eight minutes, then prepared with ether and tincture of methiolate. A longitudinal paramedian incision 10.5 cm. long was made through the skin, subcutaneous tissue and anterior sheath of the rectus muscle. The rectus muscle fibers were separated. Three cubic centimeters of a six-hour culture of *Staphylococcus aureus* was introduced posterior to the muscle, then the rectus muscle and anterior sheath were closed using interrupted silk sutures. The skin was sutured with the same material. A second group of eight dogs was scrubbed for eight minutes, 96, 72, 48 and 24 hours before operation and the experiment was repeated.

These experiments simply corroborated previous observations except that only 50 per cent of the controls were infected when male dogs were used. The female dogs invariably developed an extensive infection which involved breast tissue, showing severe mastitis. Fifty per cent of all scrubbed dogs, regardless of time up to 120 hours, showed wound infections. There

Fig. 2.—Photomicrographs of abdominal wounds in guinea-pigs removed on the 6th postoperative day. Five-tenths of a cubic centimeter of a pure culture of *Staphylococcus aureus* was introduced into the wound as described in the text. Section on the left was taken through the

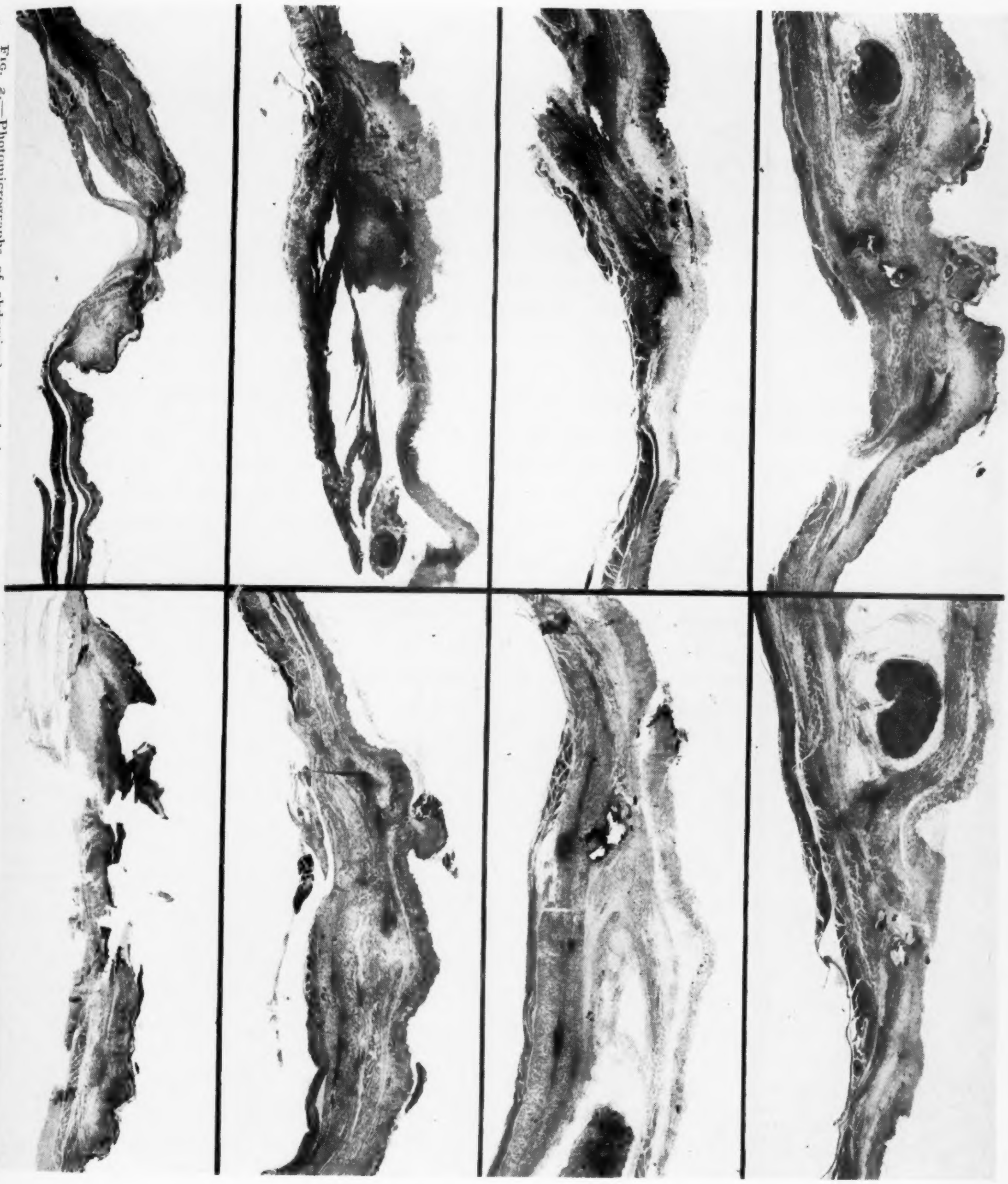

FIG. 2.—Photomicrographs of abdominal wounds in guinea-pigs removed on the 6th postoperative day. Five-tenths of a cubic centimeter of a pure culture of *Staphylococcus aureus* was introduced into the wounds, as described in the text. Sections on the left were taken from guinea-pigs which had been scrubbed 96 hours previously. Sections on the right were taken from controls of the same series. All wounds

was one death in a female dog with extensive mastitis and staphylococcus septicemia.

Twelve more dogs were operated upon, repeating the above experiment, with a study of daily temperatures and blood counts. Four animals were used as controls. The other eight were scrubbed 96, 72, 48 and 24 hours before operation. It was found that the temperature and blood counts were not reliable guides as to the presence or absence of infection. In Dog No. 231 leukocytes numbered 20,000 and 21,000 daily for four days, and yet the wound remained clean, whereas in Dog No. 233 the leukocyte count was 13,000, 11,000 and 8,000, yet his wound was grossly infected. This observation fits in fairly well with that observed clinically, for patients do not as a rule run a high temperature or leukocytosis with superficial wound infections. This is probably due to the fact that the antigen is not retained under pressure, due to looseness of tissue or early drainage. When the eight dogs that had been scrubbed were compared with the normal controls, it was impossible to determine which had had the previous scrubbing. About one-half of the animals were infected in each group.

Another study upon the effects of scrubbing in dogs was made as follows: Four animals were selected as controls and four were prepared by scrubbing 48, 24 and 12 hours before surgery. In addition, two dogs were scrubbed 96 hours before surgery and then again 24 hours before operation. In this group the 10.5 cm. incisions extended only to the anterior sheath of the rectus muscle. Also, the amount of culture introduced was calculated according to the body weight of the animal, based on the smallest amount that regularly produced wound infections without death. This was one cubic centimeter of culture for each 4.5 kilograms of body weight. Blood counts and daily temperatures were recorded. As in previous experiments it was impossible to identify those dogs that had been scrubbed from the normal controls, either grossly or microscopically. We may conclude from these experiments that the size of the dose had little to do with the resulting infections.

DISCUSSION

Since the antigen employed in our experiments was standard, infection could only occur as a result of change in the resistance of the inoculated animals. Scrubbing the abdominal wall for five to eight minutes with soap and water apparently does not greatly alter the resistance of animals to standard doses of staphylococci. However, it should be noted that guinea-pigs scrubbed 24 hours before operation showed a higher percentage of infection than did controls. Perhaps, this is due to a lowered resistance[2] which may last 24 hours or longer after trauma. It is also significant that in guinea-pigs the incidence of infection was less in those which had been scrubbed four or five days before operation. This may be due to the development of increased local tissue resistance from the trauma of scrubbing following the lag-period described by Du Mortier, or the increase in leuko-

cytes may be a factor.[2] Grossly, the difference was striking, and we thought that this method of scrubbing protected 80 per cent of the animals against infection. This observation was not borne out by microscopic examination of the wounds. Many surgical wounds are described as clean in patients, which on microscopic examination would no doubt reveal subclinical infections as commonly as observed in the experimental animal.

Clinically, there is often an increased amount of wound bleeding in abdominal incisions made in the presence of active intraperitoneal infection. We sought to reverse this phenomenon and see its effect upon intraperitoneal immunity. No demonstrable reaction or increase in resistance to intraperitoneal contamination could be demonstrated by abdominal scrubbing.

It must be admitted that the scrubbing as ordinarily practiced clinically is merely a cleansing process and is in no way comparable to the surgical scrubbing of our experimental animals. In order to test the efficacy of this method of surgical preparation we selected a group of patients with intestinal fistulae, for in this group there was obvious contamination of the abdominal wall. These cases will be published in a supplementary report.

CONCLUSIONS

1. The body temperature and leukocyte counts in experimental animals with surgical wound infections are not reliable guides for the detection of such infections.

2. High environmental temperatures in experimental animals retard wound healing.

3. Surgical scrubbing of the abdominal wall with soap and water 24, 48 and 72 hours before operation does not protect animals against wound infection. It may predispose to a decreased resistance.

4. Scrubbing 96-120 hours before surgery seems to increase resistance slightly in guinea-pigs, probably due to the stimulation of the blood supply. There is a significant increase in the number of leukocytes immediately after surgery in this group.

5. In dogs, resistance to infection was not influenced by preoperative scrubbing, regardless of the time interval before operation or of the dose of *Staphylococcus aureus* used.

6. Scrubbing the abdominal wall does not affect the peritoneal resistance.

The authors wish to gratefully acknowledge the assistance of Dr. Lyle Weed, of the Department of Bacteriology, Indiana University School of Medicine.

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SYMPOSIUM ON PROBLEMS RELATING TO LAW AND SURGERY

THE LATE EFFECTS OF CRANIOCEREBRAL INJURIES

A CONSIDERATION OF THE CRITERIA NECESSARY TO EVALUATE THE POSSIBLE CAUSES

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PRACTICALLY every member of the medical profession and many lawyers have had occasion from time to time to try to estimate the significance of symptoms which were the subject of complaint by the patient or client and which were allegedly caused by an antecedent blow on the head months or years previously. The difficulty and confusion that arise when attempts are made to separate the symptoms and signs that are traceable to organic as opposed to so-called functional changes is, and has been, generally recognized. This confusion is rendered more chaotic by the tendency on the part of the medical and legal professions to be careless, or grossly inaccurate, in making and accepting the original diagnosis and in estimating therapy and prognosis in terms of pathology. This is enhanced by the public's indifference to the need for such accuracy. Such examples as the almost universal use of the term "concussion" to cover all varieties of craniocerebral injuries; and the unjustified importance granted by the legal profession to the visible "fracture of the skull," which is of no significance, in comparison with the neglect of the invisible brain injury which is actually the cause of all the symptoms, are all too common. An equally usual but no less troublesome factor is the ease with which any physician who is legally entitled to practice medicine in this Commonwealth can qualify himself in our courts as an expert in a subject about which he actually knows little or nothing and in which his experience will hardly compare favorably with that of a well-trained house-officer. Because of this, the court is forced to resort to the legal subterfuge of considering the significance of such testimony in the light of the "weight of the evidence." This imposes on the judge, commissioner or jury the duty of interpreting the medical significance of the facts as presented to them in terms of the witnesses' personality, his ability to speak extemporaneously and his salesmanship instead of on the basis of his professional qualifications. It is evident that the solution of this problem is not to be taken lightly, and must await education in the need for accuracy of expression and thought when dealing with such an important medicolegal matter as injuries of the nervous system.

I have had occasion to point out the late effects exerted by the proper approach by both the doctor and the patient to the latter's convalescence.[1]

Denny-Brown,[2] Symonds,[3] and Jefferson, Cairns and Brain[4] have studied the end-results of craniocerebral injuries among a certain group of casualties in the British Army. Cairns,[5] Symonds,[6] the author, and many others, have written on various aspects of the surgical and early diagnostic phases of craniocerebral injuries. Much of this work is not germane to the present discussion however. That by Denny-Brown,[2] and Symonds[3] is the most pertinent but because their conclusions are drawn from a study of British Army casualties, and comprise a special group of men living and treated medically under special conditions, who are all possessed of a common language and a great emotional urge to return to their original jobs if at all possible, their conclusions may not apply at all in the study of a group of injured drawn by-and-large from American wage-earners, and, in any event, should not be considered usable without further confirmation. The standards of living and the efficiency of medical care in these latter are as varied as their different languages and customs. If such patients have any emotional urge to return to their former jobs it is in spite of the influence of their friends, families and lawyers, and is influenced, moreover, by the effect on their morale of getting something—that is compensation—for nothing, which is the privilege of not working for as long as they can produce sufficiently disabling symptoms.

This is perhaps an oversimplification of an extraordinarily complex question as far as estimating the genuineness of the patient's symptoms is concerned. The impact of even a minor craniocerebral injury on the personality of a workman who is constitutionally unable to hold a job or who is so emotionally unstable that his family life is continually disrupted, will be decidedly different from that of the same injury on the personality of an emotionally stable, intellectually competent, adequate individual. If this comparison is carried either to the extreme of no injury plus a psychotic or psychopathic personality, or of a maximum injury to a completely normal personality, and the other possible combinations and their effects and interplay imagined, the importance and magnitude of this one aspect of this problem becomes immediately evident. If to this are added the varying influences exerted by the accuracy of diagnosis, the efficiency of treatment, the potentiality for harm that lies in an uneducated public opinion, in insurance adjusters, lawyers, friends, members of the family and other interested advisors; the amount and severity of any permanent residual loss of function; the influence of recognized or unrecognized intercurrent diseases, advancing age, intentional or unintentional malingering, impairment of judgment, and the like, on the part of the patient, some slight conception of the need for clarification of this matter can be had.

As far as I know, there are no reliable or worth while data on the effect of the relationship between a craniocerebral injury and the pretraumatic personality of the injured. This phase of the larger problem must await further and more detailed study of patients in both the acute and late

stages of their injuries. No attempt will be made to deal with it here. I do however, feel that there are available certain other data relative to the diagnostic criteria that must and can be met if the symptoms alleged to have been originally caused by an antecedent craniocerebral injury are to be properly evaluated, a diagnosis made and therapy recommended at a late date. These criteria are tentative and will doubtless be modified many times as experience with them accumulates. The chief justification for bringing them to the attention of the medical and legal professions is that at least a start will have been made toward the solution of this vexing matter. They are based upon experience extending over a number of years, and are exemplified here by statistics drawn from a group of 50 patients injured in industry and seen and studied by myself during the past 18 months as an impartial examiner for the Massachusetts Industrial Accident Board. Such a small series justifies only the grossest statistical conclusions, but until many hundreds of such patients have been studied and tabulated there is reason to believe that it is as accurate as a somewhat larger but still inadequate series would be. It has the additional advantage of unity, in that all are workman's compensation cases, and of impartiality, in that the data have neither been collected in the interest of the complainant nor of the respondent.

This series includes 8 female and 42 male patients. The age was known in 39 of the 50 (32 males and 7 females). The oldest patient was 71 and the youngest 18, both being males. Similar figures for the women were 44 and 19, respectively. The interval between the date of the injury and the time of the first examination was known in 49 instances. This was considered in relation to the diagnosis as determined at this examination. The diagnostic classifications, which are discussed in more detail below, were divided under five headings: *Organic,* in which the cause of the symptoms was believed to be organic and certainly related directly to the alleged accident; *posttraumatic-state,* in which the cause of the symptoms was believed to lie in the nonorganic spheres and to be not necessarily directly related to the alleged accident; *intercurrent disease* and *no disease,* in the first of which the cause of the symptoms was believed to lie in an intercurrent disease having no relationship to the alleged accident, and, in the second of which, the symptoms and signs were of such character that it was believed that there was no disease of any kind present at the time of the examination; and, finally, *double diagnosis* in which two diseased conditions were believed to be simultaneously present. The average interval between the receipt of the injury and the first of my examinations in the organic group, was 14.5 months; in the posttraumatic-state group 12.5 months; in the double diagnosis group 13 months; in the intercurrent disease group 14.25 months, and in the no disease group 8 months. The longest intervals were 31 months in the organic and 30 months in the posttraumatic groups. The shortest intervals were 2 months in each of the same two groups.

There were 15 patients in the organic, 18 in the posttraumatic-state, 6 in both the no disease and intercurrent disease, and 4 in the other group. Seven patients were examined a second time, and in 1 patient the classification could not be determined. This patient reported for examination 10.5 years after the injury that was alleged to have caused his symptoms. No data were collected in regard to the marital state or the kind of work that was being done at the time the alleged injury is said to have occurred. I do not believe that, with the possible exception of a tendency on the part of the patients classed in the no disease group to report, on the average, twice as quickly for examination after they have recovered from their acute episode as those in the other groups, these data have any diagnostic or prognostic significance. Even the exception noted cannot be taken too seriously because the time when the examination was performed was conditioned by such factors as the diligence of the patient's lawyer, the resistance of the insurer's counsel and the state of the docket of the Industrial Accident Board. These data are included only because if they were not it would inevitably be suggested that their omission was significant.

VERIFICATION OF THE INJURY

The criteria sufficient to justify a conclusion that the symptoms and signs complained of by the patient at a late date after an antecedent accident are both positive and negative and action based on such conclusions—whether therapeutic or legal—are weakened by just that much if and when either class is not investigated. Among the fundamental information that must be obtained in every case before anything further can be decided is whether the patient has actually sustained a blow on the head, and, if so, whether there is reason to believe it was sufficiently severe to produce resultant changes in the brain, and, finally, whether the significant blow, if any, actually did occur at the time claimed. This last consideration can be disposed of first. It is more a matter of judicial than medical investigation and the doctor should ordinarily accept the patient's statement in this regard pending correction or verification by proper authority. He should avoid putting himself in the position of an insurance or similar investigator and leave that phase of the problem to those whose opportunities and training equip them to do it properly. Assessment of responsibility among repeated craniocerebral injuries will occasionally be necessary of course, but no general rule can be laid down for these occasions. Each is individual and will necessitate making available to the physician all the material that is germane to the problem before he can even approximate a decision. The verification of the date of the accident did not arise in this series of 50 patients. Judicial action by the Industrial Accident Board had identified the time and place of accident in every instance. There was a history of multiple craniocerebral injuries in only one case. No difficulty was met with in establishing the sig-

nificant accident—which proved to be the second—because the patient volunteered the information that he had been completely well and doing full-time work in the interval between the two. He was classed in the posttraumatic-state group because his symptoms were believed to be nonorganic in origin.

LOCAL OBJECTIVE EVIDENCE OF THE INJURY

To determine whether or not the patient has actually struck or been struck on his head may at times be very difficult. This will be especially true in the absence of visible or palpable signs and may involve check interviews with witnesses that one would not ordinarily see. Scars, and the like, on the scalp will be indicative, but not necessarily conclusive, evidence of this fact, inasmuch as the age of a completely organized scar cannot be accurately estimated and certain patients may use the scar from a previous accident to bolster their claims for damages on account of a later injury. Positive and negative roentgenograms of the skull must also be accepted with caution as final evidence for or against injury sustained at a given time. In the first place, my experience leads me to believe that in a disputed case certain differentiation of a fracture line from a blood vessel marking or suture line cannot be made without stereoscopic lateral roentgenograms, at the very least. Furthermore, to demonstrate such a differentiation, the films must be viewed in a stereoscope and not one-by-one in their flat state. Further confusion is caused by the fact that the rate and amount of bony union of linear fractures of the skull are completely unpredictable. It is true that, in general, such fractures that occur in children and young adults tend to heal quickly—in a matter of weeks—whereas similar fractures in the skulls of the elderly tend never to heal by bony union. Thus, in the first instance a roentgenogram that is positive at the time of injury may be negative when repeated at the time the patient is to appear in court. Presentation in evidence of only the latter film, with the suggestion that because it was negative the former was misread, would not, necessarily, be in accord with the truth, and might work a grave injustice on the complainant. On the other hand, it is possible to do the respondent an injustice also if the suggestion, that because a linear fracture remains visible roentgenologically after months or years, and hence is evidence in itself of a continuing cause of symptoms, is not branded as false and without basis in fact. In this group of 50 patients 25 showed positive evidence of head injury at the time claimed, in the form of the scar of a scalp wound or the demonstration of a fracture of the skull roentgenologically. Of these 25, ten were in patients whose symptoms were believed to be on an organic basis, and traceable to the injury, nine were in those with symptoms on a posttraumatic-state or nonorganic basis, two occurred in patients with a double diagnosis, two were noted in patients in whom no disease could be demonstrated, and in one the symptoms found at the time of the examination were believed to have been caused by an

intercurrent disease. In contradistinction to this, five of the organic, nine of the posttraumatic-state, two with a double diagnosis, four with no disease, and five with an intercurrent disease showed no evidence of either a scalp wound or fracture, and any conclusions as to the actuality of the blow in these patients had to be drawn from other evidence. Insofar as these figures go, and this is borne out by my experience in other cases, one may conclude that a scalp wound or properly identified fracture of the skull is good, but neither essential nor conclusive, evidence in support of a claim that present symptoms have a background of a preexisting blow on the head. In the absence of such a demonstration, recourse must be had to the history and to other objective physical findings of trouble with the central nervous system. In this connection, it seems probable that if the patient's symptoms, as complained of at the time of the examination, are traceable either to an intercurrent disease or an organic as opposed to a psychogenic cause, there will be found at examination to be positive objective physical signs of such disease or injury.

THE INTRACRANIAL EFFECT OF THE INJURY

The next decision that has to be reached, once the examiner is convinced that the claimant actually did strike his head, is whether or not the blow was sufficiently severe to produce intracranial pathology. Intracranial rather than cranial pathology is specified because any damage to the skull that does not simultaneously, or later, affect the skull contents—with the exception of the rare unrecognized compound fracture that becomes septic—will not produce late symptoms that have any direct cause and effect relationship to the alleged injury. Evidence that justifies the opinion that there has been such significant cerebral damage inflicted at the time of the injury, and that, therefore, the present symptoms can be correlated in a cause and effect relation to that damage is, for all practical purposes, concerned with the demonstration of loss of consciousness as the direct result of, and at the time of, the receipt of the alleged blow. It is true, as Denny-Brown and Russell[8] have shown experimentally, that loss of consciousness is traceable to acceleration and/or deceleration of the head, and that, therefore, it is possible to visualize a situation in which lethal brain damage might be caused without such loss. In support of this view, Denny-Brown[9] has instanced slow crushing of the head and penetrating wounds of the skull and brain by small missiles, usually multiple, and with very little inherent energy, either of which type of injury can and has caused sufficient brain damage to serve as the origin of late symptoms. I, too, have observed such a sequence of events following both a crush and stab wounds of the skull. When these latter accidents occur, however, there is no doubt in anybody's mind relative to the actuality of such an alleged fact, and confirmation of the significance of the intracranial damage is not necessary by other means. Penetrating wounds made by small low-energy fragments, such as follow bomb or similar explosions, are not ordinarily seen in the absence of a war, and

are easily recognized from the history as a possible cause for the complainant's symptoms. The only other source of error arises from the presence of a solid subdural hematoma resulting from the rupture of a bridging vein. The injury causing this may be so slight as not to be accompanied by unconsciousness. The clot in these patients will produce symptoms which vary from a mild but persistent headache to those usually associated with large brain tumors. A differential diagnosis between such a clot and a brain tumor is impossible without operative visualization. Patients with these symptoms and signs will usually prove to be such baffling problems that hospitalization, with a diagnostic study by all the means at hand, including pneumo-encephalogram, will prove necessary before the physician is justified in expressing an opinion relative to the cause-and-effect relationship between the accident and alleged resultant symptoms. Fortunately, these clots are rare—indeed the rarest of all subdural hematomata—and, therefore, one of the rarest of the intracranial complications of head injury. I[10] have shown that chronic solid subdural hematomata occurred only 45 times in a series of 310 verified cases of all types of such clots. Furthermore, in only three of these 45 clots could a diagnosis of "brain injury" be made or a fracture of the skull demonstrated. Since the estimated occurrence of all subdural hematomata varies from one per cent (Browder, *et al.*[11]), to my[10] estimate of ten per cent, and since the occurrence of this particular solid type of clot is only 14 per cent of that, it is evident that the mathematical chance of finding one of these lesions as the cause of the claimant's symptoms is very small indeed.

LOSS OF CONSCIOUSNESS AND AMNESIA

Consciousness and, hence, unconsciousness are difficult to define satisfactorily. Seven types have recently been described by Miller,[12] who also gives 16 definitions of the word. I prefer Cobb's[13] definition, in which consciousness is described as awareness of, and intellectual contact with, the individual's surroundings. This is highly variable from person-to-person and from time-to-time in any one person, and it is probable that, in the last analysis, no person is ever wholly conscious. Loss of consciousness, moreover, is not a definite entity as to time or amount. It merely implies a decreased awareness of his surroundings by the individual suffering from this phenomenon. This may be described in such terms as "dazed," "fainted," "groggy," "woozy," or "dizzy," to indicate the short periods, and as all the varieties of coma to indicate the longer ones. The demonstration of such a decrease in the patient's awareness of his surroundings may be extremely difficult and tax the ingenuity of the examiner to the utmost. One of the more reliable methods of verifying the presence of such a phenomenon is to investigate the patient's loss of memory in its relationship to the period before, during and after the accident. Retrograde amnesia—that is, a loss of memory for events that preceded the accident—especially if it is associated with accidental and anterograde amnesia is, in my experience, pathognomonic

of a loss of consciousness which is attributable to the accident. It cannot be confused with a similar condition caused by syncope, and offers opportunities for factual verification that will expose the malingerer. Amnesia that includes the time during which the accident was taking place and a period of time following it, but not necessarily including the actual start of the fall or receipt of the blow, is presumptive evidence of loss of consciousness. Postaccidental amnesia alone is, of itself, no evidence for or against loss of consciousness when it is not associated with either retrograde or accidental amnesia. Its signficance depends upon its relationship to other factors in the case. The examiner should not be confused or jump to wrong conclusions because there may be a period immediately following the accident during which the patient talks and acts normally or because the patient has "islands of consciousness" in the sea of unconsciousness. Neither of these phenomena alter the diagnostic or prognostic significance of the unconscious period. I have seen an airplane pilot who crashed with a "dead stick" from 5000 feet. When rescued he was stepping out of the plane. He was taken by automobile five miles to a hospital, and during this trip told in detail how he had landed his ship without killing himself. On arrival at the hospital he became comatose, and for four days responded only to painful stimuli. At the end of that time he was again mentally normal, conscious, and in full contact with his environment. He was then asked to redescribe the landing of his plane but was unable to do so, and had no memory of anything from the time his engine failed at 5000 feet to the time he regained touch with his environment four days later. He was shown to have suffered a fractured skull, lacerated and contused wounds of the scalp, a contused brain, and other less serious injuries. Squadron Leader H. L. Burton,[14] of the Royal Air Force, has also described this sequence of events as of sufficient importance to justify the medical personnel of the Royal Air Force, when investigating injuries sustained by pilots in "crashes" or by the riders of motorcycles in collisions, in obtaining the story of the loss of consciousness not only at once after the accident but some hours later as well. In this group of 50 patients (Table I) 33 had a history of loss of

TABLE I

UNCONSCIOUSNESS AND AMNESIA

		Organic	Post-traumatic-State	Inter-current Disease	No Disease	Double Diagnosis	?	Totals
Unconsciousness	Yes	12	11	0	5	4	1	33
	No	0	5	6	1	0	0	12
	?	3	2	0	0	0	0	5
Amnesia	None	0	6	6	3	0	0	15
	Retrograde	0	5	0	0	0	1	6
	Postaccidental	12	11	0	3	4	1	31
	?	3	1	0	0	0	0	4

consciousness, 12 had no loss of consciousness, and in five there was doubt as to this fact. Among those shown to have been unconscious, 12 had symptoms that were classed as organic, those of 11 were considered to be non-

organic, those of four were traceable to the combination of two conditions, and in five it was felt that no disease was present when they were seen later. Five of the posttraumatic-state group, six with intercurrent, and one with no disease, were considered not to have been unconscious. In regard to the incidence of the various kinds of amnesia there were 12 patients with postaccidental, three who might or might not have had any kind of amnesia, and none who did not have some loss of memory among the "organic" group. Language difficulties accounted for the three undetermined cases. Among those whose symptoms were classed as posttraumatic-state or on a nonorganic basis, five had retrograde, 11 had postaccidental, one could not be determined, and six had no amnesia. Four patients who had a combination of organic symptoms and intercurrent disease as a basis for their symptoms had postaccidental amnesia, six who were suffering from an intercurrent disease alone had no amnesia, six that had no disease were equally divided between postaccidental and no amnesia, and one patient, that could not be classified, had a combination of retrograde and postaccidental loss of memory, as did five of the posttraumatic-state group already listed. There was no evidence of any original intracranial injury in any of the patients in the intercurrent disease group. It seems reasonable to conclude that unconsciousness at the time of the injury predicates intracranial pathology sufficient to serve as an adequate cause for the production of late symptoms. On the other hand lack of unconsciousness at the time of the original injury can be taken to indicate that any late symptoms are almost certainly either traceable to a functional nonorganic upset or are caused by an intercurrent disease. All patients who were certainly known to have been rendered unconscious originally, gave a history later on of some type of amnesia, which was usually postaccidental.

CLASSIFICATION OF THE SIGNS AND SYMPTOMS OF LATE DISABILITY

Having verified, or failed to verify, the claimant's allegation that he was struck on the head at the time of the alleged accident sufficiently hard to cause intracranial damage, one must now make some attempt to tabulate and classify the myriad symptomatology that the patient is only too willing to describe. Care should be taken never to ask direct questions or to otherwise suggest by naming it any particular symptom. All these patients are highly suggestible and ready to adopt as their own almost any symptom that is brought to their attention. I have never believed that there is any special diagnostic value to be found in the characteristics of purely subjective symptoms. Objective signs however, even if described as symptoms, are different. Some demonstrable pathology can be found behind such symptoms in practically every instance if the examination be sufficiently detailed and searching. This is not to be taken to mean that subjective symptoms are without significance. It is merely that that significance must be interpreted in the light of other and objective data, whereas objective signs have their interpretation inherent in them. In addition to the abnormalities found in physical and neurologic examination, the 50 patients in this group com-

plained voluntarily of 17 symptoms. Because the number of other such possibilities is practically legion only these 17 will be considered here. They are fairly representative of the larger group, and I believe that conclusions reached from their analysis will hold true for the rest.

OBJECTIVE SYMPTOMATOLOGY

If the abnormalities found in physical and neurologic examinations are excluded, four such symptoms fall in the objective group (Table II). These are convulsions; loss of memory or change in personality; paralysis of one or more extremities; and loss of weight. In addition, physical abnormalities demonstrated at general physical and neurologic examinations, and excluding scars of scalp wounds or fractures of the skull, were present in 13 of 15 in the organic, both patients who were classed as belonging in the double diagnosis group (a combination of organic and intercurrent disease), and in all five of the patients with intercurrent disease. Two organic cases,

TABLE II

OBJECTIVE SIGNS AND SYMPTOMS

	Organic	Post-traumatic-State	Inter-current Disease	No Disease	Double Diagnosis	?	Totals
Scalp wound or tracture Yes	10	9	1	2	2	1	25
Scalp wound or tracture No	5	9	5	4	2	0	25
Other physical signs Yes	13	1	5	0	2	1	22
Other physical signs No	3	17	0	6	2	0	28
No physical signs at all	0	9	1	1	0	0	11
Convulsions	4	1	2	0	?1	1	9
Loss of memory or personality change	4	4	0	0	1	1	10
Paralysis of extremity	0	0	1	0	1	0	2
Loss of weight	0	3	1	0	0	0	4

who had no abnormal physical signs, had convulsive seizures that developed as the direct result of the earlier injury. The two patients with no disease, and nine out of 18 patients in the posttraumatic-state group had no abnormal physical signs. Convulsions were present nine times—four among the organic, two in the intercurrent disease group, and one each in the posttraumatic, double diagnosis and unclassified groups. Loss of memory or change in personality occurred 10 times—four each in the organic and posttraumatic-state, once in the double diagnosis, and once in the unclassified groups. Three patients who were reexamined were found to have a change in personality at the second but not at the first examination. Two of these were in the organic, and one in the intercurrent disease group. While it is unfortunately true that although the disability claimed by a patient as the late result of an antecedent accident need not result from an objective physical defect, the very fact that such a defect is present puts the burden of proof on the examiner to show that it is not the causative factor. No greater significance than that can be attached to the demonstration of abnormal physical signs and what may be spoken of as objective symptomatology in a consideration of the relationship between an antecedent craniocerebral injury and later disability.

SUBJECTIVE SYMPTOMATOLOGY

Subjective symptomatology that is allegedly the cause of late disability in craniocerebral injuries is the most difficult part of this whole problem to evaluate. The possible causes of headache, insomnia, dizziness when stooping, general pains, and the like, are legion. There is no present evidence to show what the statistical chance of their occurrence is, in either normal persons or in those afflicted with the various cerebral changes that follow craniocerebral injuries. A patient with a known psychopathic personality, and an imaginary craniocerebral injury, will, and often does, complain of the same symptoms and the same disability that another with a fluid subdural hematoma following a severe injury, or still another with generalized cerebral atrophy that has followed a mild injury in an arteriosclerotic does. To separate each from the others will require a knowledge of the pretraumatic personality of the patient, a detailed study of his personality and state of physical health at the time of the complaint, an objectively accurate diagnosis of the pathology present at the time of the injury, and in the case of the atrophy a pneumo-encephalogram, and in that of the subdural fluid hematoma an exploratory operation before the differential diagnosis can be made, and the cause-and-effect relationship between the accident and the disability unravelled. Even this may not give the full explanation because, for example, in the hematoma case an associated neurosis may still persist after the correction of the physical abnormality. When such symptoms remain a demand will be made on the doctor to divide the responsibility for this continued disability between a possible instability of the patient's pretraumatic personality, the changes induced by an unfavorable environment before or after the injury and the still persisting effects of an intracranial abnormality that may have been present for some weeks or months.[15] Other even more complicated relationships can be easily visualized, and the hopelessness of reaching a just conclusion relative to the importance of such subjective symptomatology as a cause of late disability after previous craniocerebral injuries can be realized. Much psychometric, social service, psychiatric, psychologic, neurologic, pathologic and surgical data relative to the acute as well as the more prolonged effects of all varieties of craniocerebral injuries must be collected before the significance of this class of late symptoms can be evaluated or even recognized as existing. Headache, dizziness on stooping, and general weakness were the commonest of this class of symptoms in this group of 50 patients (Table III). Forty-four of the 50 had headaches, 28 dizziness, and 16 general weakness. Of the six patients who had no headaches, one each out of 15, and 18, respectively, were in the organic and posttraumatic-state, and two each out of six in both the intercurrent and no disease groups. Thirteen of the 15 organic, eight of the 18 posttraumatic-state, one-half of the intercurrent disease and double diagnosis, and one-third of the no disease groups complained of dizziness on stooping. Seven of the organic, five of the posttraumatic, two of the intercurrent disease,

and one each of the no disease and double diagnosis groups, complained of generalized weakness. Nervousness (13 times), and insomnia (12 times), were the next most common. They were as common in the organic as they were in the posttraumatic-state group. The frequency and classification of the rest is noted in Table III. The English school[2, 3, 4, 5, 6, 8, 9] has shown that before conclusions could be drawn relative to the relationship between such symptoms and their antecedent accidents, as occurred among casualties in the Royal Air Force, information relative to the patient's personality, home life, relation to environment, work record, intellectual level, and the like, had all to be collected for both the time preceding and that succeeding the accident. This was possible without undue expenditure of time, money and personnel in this British group. It would not be possible in the ordinary industrial or automobile accident case in this country. However, that does not imply that such investigation should be altogether neglected. As much of this information as is possible should be acquired in every patient. A

TABLE III
SUBJECTIVE SYMPTOMS

	Organic	Post-traumatic-State	Inter-current Disease	No Disease	Double Diagnosis	?	Totals
Headache Yes	14	17	4	4	4	1	44
Headache No	1	1	2	2	0	0	6
Dizziness on stooping	13	8	3	2	2		28
Weakness	7	5	2	1	1	0	16
Nervousness	6	4	1	1	0	1	13
Insomnia	5	4	1	2	0	0	12
Pressure in the head	5	2	1	1	1	0	10
Inability to concentrate	5	2	1	1	0	0	9
Noise in the ears	6	2	1	0	0	0	9
Difficulty with sight	3	4	0	0	0	0	7
Photophobia	2	1	0	0	0	0	3
Increase of symptoms in wet weather	1	1	1	0	0	0	3
Intolerance of noise	2	1	0	0	0	0	3
Confusion in crowds	1	1	0	0	0	0	2

family history of insanity, epilepsy, sick headaches or conditions similar to the one from which the patient suffers, evidences of marital incompatibility, addiction to alcohol or drugs either in the family or on the part of the patient, will help clarify the patient's emotional background and permit an estimate of his pretraumatic personality to be made. His work record, including the frequency with which he changes jobs, the number of his promotions and the reasons or lack of reasons there-for, give further insight. A review of his educational background, a knowledge of the country from which he or his family originated, and observations on his emotional reactions while undergoing examination, are all additional helpful data that can be obtained with little or no extended investigation. Finally, such evidences of an oversensitive sympathetic nervous system as cold and discolored extremities, excessive sweating, fainting, irregular pulse, dermographia, "goose-flesh," and the like, may well provide the clue to otherwise unexplainable symptomatology and permit recognition of the fact that the disability claimed arises not from the antecedent accident but rather from emotional or psychic deficiencies in-

herent in the victim. All such data as can be obtained in the present state of our knowledge from a study of the above and other subjective symptoms, is to the effect that when any one of such symptoms acts, of itself, as the chief cause of late disability following an earlier craniocerebral injury it is of no value either for diagnostic purposes or for determining the cause-and-effect relationship between the accident and the later disability. The only positive virtue that its presence affords is in forcing the physician to use all possible methods of diagnostic investigation if he would really determine the reason for and the significance of it.

EFFECT OF EARLIER THERAPY

One other possible variant must be briefly mentioned: Its effect is impossible to estimate and yet is a real factor in the prolongation of disability and in its influence on the patient's morale and later subjective symptomatic reactions. It is the efficiency of the treatment rendered at the time of the accident. As was pointed out in the first section of this paper, this is intimately connected with the accuracy of the original diagnosis. A subdural hematoma that is promptly recognized and removed will cause less permanent cerebral changes and, hence, less later disability than one that is not recognized and removed for some months and until the late disability has already developed. A lacerated brain that is diagnosed and treated as a case of concussion falls into the same category. Moreover, a true case of concussion that is forced to undergo a prolonged convalescence because the doctor has mistakenly considered it a "serious brain injury" has the cards stacked against him, and will inevitably develop a neurosis and disabling symptoms sufficient in number to justify to himself, his friends and family the fact that, although acting and feeling well, he is nevertheless an invalid. In only one of the 50 cases reported herewith, was it possible to state that sufficient objective data had been collected at the time of the accident to justify making a diagnosis in terms of pathology. As an inevitable result the immediate therapy left much to be desired and was usually conspicuous by its absence. Even those patients who had no disease at the time of their examination were well in spite of, and not because of, the therapy they had received earlier. The length and psychologic handling of the convalescence may also prove to be important factors in influencing the occurrence of later symptoms. I[1] have dealt with this elsewhere, and will only repeat that: "The convalescent care of patients with craniocerebral injuries should not be of a hit-or-miss type but must be carefully planned to keep the inevitable associated destruction of initiative and self-confidence from developing into a permanent neurosis, and spoiling the end-result of what might, otherwise, have been a successful case." In two of the patients in this series of 50, the symptoms and disability arising therefrom were unquestionably traceable in one case to a convalescence that was too prolonged and inactive, and in the other to one that was too short and active. Eleven of the 15 organic, 12 of the 18 posttraumatic, five of the six intercurrent

disease, and two of the four double diagnosis cases, were doing, and had done, since their injury no work and taken no exercise. One organic and one posttraumatic case worked about the house, and another of the latter group took moderate exercise. All the patients with no disease, three of the organic, four of the posttraumatic, the other two of the double diagnosis group, and one unclassified case were at work when seen.

THE INFLUENCE OF PENDING LITIGATION

The influence of pending litigation on the persistence of symptoms in these patients is a real but rather overstressed factor. The causes are obvious, and their elimination equally so. Four of the patients in this group of 50 had a coincidental complete recovery from their disability and the ending of litigation by settlement of their claims. Attention should be called to the fact that although such a coincidence is presumably due to something analogous to malingering it is not necessarily so. A patient with neurosis based upon dread of appearing in court will react similarly to one who fears injustice because of his inability to demonstrate the truth of his claim in the same surroundings. In such circumstances, these patients, like the malingerer, will recover from their disabilities with the ending of their litigation.

CLASSIFICATION OF THE DISABILITY

Before settling the question of cause-and-effect relation between the accident and later disability the patient must be classified as to the type of his disability. I have found it convenient to do this under five headings. These have been referred to previously in the body of this paper and are as follows: *Organic; posttraumatic-state; intercurrent disease; no disease* and *double diagnosis.*

In the *organic group* are classed those patients who sustained a significant blow on the head and who can be shown to have organic objective abnormalities that are not traceable to intercurrent disease, and that may occur either with or without functional subjective symptoms. The demonstration of such objective abnormalities usually will require hospitalization, often pneumoencephalography, and frequently bilateral exploratory trephination. Unless the demonstration in an office is unquestionable, the physician should not commit himself relative to this possibility without either qualifying his statements or having the benefit of such hospitalization. In this group of 50 patients there were 15 who were classed as organic. All were seen first in the office, and an unqualified diagnosis and opinion refused in 12 pending hospitalization. In most instances, for various reasons varying from the unwillingness of the patient to cooperate, to the influence of friends and financial stringency, the recommendation for hospitalization was not acceded to, and the qualified findings were accepted. It is my considered opinion that this was a mistake and should not have been permitted. Of the three for whom hospitalization was not recommended, and an unqualified diagnosis

and opinion rendered, two had been previously hospitalized, even to the extent of transtemporal exploration and in one sufficient objective evidence was amassed at the office examination to obviate further hospital study.

The *posttraumatic-state* group includes those patients who sustained a significant blow on the head but who at the time of the later examination do not have any organic objective abnormalities but do have functional subjective symptoms. The demonstration of such a lack of objective abnormalities cannot be considered final unless, and until, hospitalization with pneumo-encephalogram, and other similar tests, have proved normal. In some cases, particularly those about whom one has any doubt because of the length, persistence or disabling quality of the symptoms, bilateral exploratory trephination will have to be resorted to as well, before the doctor can make an unqualified diagnosis. It is this group of cases that includes all the most difficult problems relative to cause-and-effect relationship between the accident and disability. Often it will be impossible to be sure of this relationship and any information bearing on pretraumatic personality, personal relationships, environment, habits, illnesses, work record, *etc.,* is sure to be helpful. The family and educational history must be investigated, and a search for hereditary or obscure diseases, must also be carried out. With this, and similar information at hand, it will be possible in many cases to decide whether the claimed disability has actually been caused by the preceding accident or by an hitherto unsuspected, and perhaps inactive, effect of an abnormal pretraumatic personality or other personal peculiarities. To avoid confusion in nomenclature I have preferred to use the term posttraumatic-state for this group of symptoms. It has many other names, some without meaning, as postconcussional-state, and others, as posttraumatic neurosis, that are more descriptive than posttraumatic-state. The latter diagnoses imply too much however, and may be as harmful as the meaningless ones. In the 50 cases reported herewith there were 18 on whom this diagnosis was made. Four were first seen in the hospital and 14 in the office. Qualified diagnoses were made on five pending hospitalization. Unqualified diagnoses were made on nine others, none of whom were recommended for hospitalization. Two of these had become free of complaints and disability just prior to examination and following settlement of litigation about their claims. Two had hyperactive sympathetic nervous systems, without other symptoms. One of these was also of very low intelligence and extremely emotional. In two the symptoms were located in the region of the supra-orbital nerve but since the patients had probably been knocked unconscious by the blows which caused the bruises about their eyes, they were considered to have probably had a mild craniocerebral injury. They were included in this group despite the highly localized character of their symptoms. One other patient was alcoholic and another had a very bad work history, and, in addition, sustained her injury by jumping out of a window in an attempt to escape a fire in the factory where she worked. These were considered adequate causes

for later symptomatology. Finally, the ninth patient had had two previous hospitalizations, including exploratory trephination—all of which were negative—before I examined him.

The importance of recognizing that more than one diseased condition may be present, and that the combination may thus enhance the symptoms and disability inherent in either one cannot be overestimated. Omitting the obvious and fairly constant organic and posttraumatic-state relationship, it is in this group that the fruits of failure on the part of the employer to require preemployment and periodic physical examinations are collected. A craniocerebral injury causes enough disability when it occurs alone in a healthy individual. When the effects of a preexisting disease are added, the resulting disability may increase out of all proportion. This is especially true when the circulation is involved. Not only is the local damage produced at the time of the injury greater but treatment is less effectual. Worst of all, postinjury disability is greater, and in a large group of such patients is progressive also. Such patients are grouped under the heading *double diagnosis* which is self-explanatory. There were four patients among the 50 on whom a double diagnosis was made. All had a combination of objective organic signs, part of which were traceable to the original craniocerebral injury, and part to an associated intercurrent disease. In every case this intercurrent disease was vascular—either arteriosclerosis with hypertension or hypertension alone. One was seen originally in the hospital and the other three in the office. Hospitalization was not considered necessary in these latter because two had already been hospitalized and adequately studied elsewhere, and there was sufficient data collected from the office examination of the other to make the diagnosis unquestionable.

Patients whose disability is thought to be caused by intercurrent disease or who, at examination, have no disability are classified under the headings *intercurrent disease* and *no disease*. The first of these conditions should never be overlooked in any patient even though he himself may believe the symptoms to have been entirely caused by the original craniocerebral injury. The problem in such cases is not to recognize the fact that an intercurrent disease is present—once one remembers that possibility—but rather to decide whether the individual patient belongs in this grouping rather than in the double diagnosis classification. In the former the disability will be independent of, and in no way influenced by, any preexisting craniocerebral injury. In the latter, as pointed out above, the two are interdependent. Each such problem is an individual one and subject to no general rules for its solution. There were six such patients in this group. One was first seen in the hospital, two were advised to enter the hospital for confirmation of their diagnoses, and three presented such typical findings in the office that hospital examination was not considered necessary.

Patients classed as having *no disease* are usually those who want permission to return to work, or whose counsel want their client's medical con-

dition verified before reaching a final settlement of the litigation. The reasons for the classification are self-explanatory.

CRITERIA NECESSARY TO LINK ANTECEDENT CRANIOCEREBRAL INJURY AND LATER DISABILITY

It must now be apparent that certain criteria have to be met before the doctor is justified in giving, or the lawyer in accepting, an opinion relative to the relationship between a craniocerebral injury and later an allegedly consequential disability. Such an opinion can be unqualified only after the patient has submitted himself to an investigation that will demonstrate the significance of all objective findings and will give proper consideration to all the inherited, personal, social, educational and occupational influences that have been brought to bear upon his personality both before and after the alleged injury. If these requirements cannot be, or are not, satisfied then the opinion referred to above must be qualified appropriately.

If the opinion is to be sustained that a direct cause-and-effect relationship exists between the injury and the later disability, the following criteria must be met: 1. That a patient actually did sustain a blow on the head at the time and place claimed. 2. That that blow was sufficiently significant to produce intracranial pathology at the time of its infliction. 3. That the disability be manifested by organic signs and symptoms that could only arise from earlier and continuing intracranial pathology. 4. That the disability be manifested by a combination of organic signs and symptoms that could only arise from intracranial pathology, and the signs and symptoms of intercurrent disease or the subjective symptoms seen in the so-called post-traumatic-state. 5. That in the absence of objective signs any subjective symptoms complained of be shown after adequate investigation not to be, or have been, caused by any characteristics found in the patient's pretraumatic personality, and its changes, or in his inheritance or his social, family, intellectual, emotional, or work history.

If the opinion is to be sustained that there is no direct or indirect cause and effect relationship between the claimed injury and the later disability the following criteria must be met: 1. That the patient may or may not have sustained a blow on the head at the time and place claimed. 2. That if such a blow was sustained it was not sufficiently significant to produce any intracranial pathology at the time of its infliction. 3. That there be no organic signs or symptoms found after a sufficient search which could reasonably be supposed to arise from an earlier and continuing intracranial pathology. 4. That any organic signs or symptoms that are present be most reasonably explained on the basis of an intercurrent disease. 5. That any subjective symptoms present be most reasonably explained on the basis of characteristics found in the patient's pretraumatic personality, and its changes, or in his inheritance or his social, family, intellectual, emotional, or work history.

It is the function of the court, or of an insurance or similar investigator, to verify the time and place of the alleged injury. Scars of scalp wounds,

bruises and properly checked skull fractures can verify its actuality. The demonstration of a period of unconsciousness verified by amnesia before, during or after the injury, or the presence of a solid cerebral subdural hematoma, perforating wound or wounds of the skull and brain, or a crushed skull, will verify the significance of this injury. The classification of the signs and symptoms into those caused by organic changes that have originated in either the injury or an intercurrent disease, or both, and those that are purely subjective and are either traceable to the functional as opposed to the organic effects of the injury or to characteristics found in the patient's pretraumatic personality and its changes, or in his inheritance or his social, family, intellectual, emotional, or work history, will permit a reasonably accurate estimate of the cause-and-effect relationship between the alleged craniocerebral injury and any succeeding disability.

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MECHANISMS OF HEAD INJURY

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INJURIES OF THE HEAD probably account for more deaths leading to litigation of one kind or another than does any other one category of mechanical trauma. Not only are such injuries responsible for a large proportion of all deaths by violence but they are also a common cause of nonfatal disability. When indemnification is claimed or assault is charged on a basis of such an injury, it is obviously desirable that the medical witness understands the various mechanisms by which physical forces may result in damage to the head and its contents.

Although mechanical injuries of the head may be produced in many different ways they are due, in the last analysis, to the application of force in such a manner that it changes or tends to change the state of rest or uniform motion of the structures affected. Thus, the impact responsible for injury may result from a collision between a stationary head and a moving object, a stationary object and a moving head, or a moving head and a moving object when either the direction or the rate of motion of the two is different. The site of the collision may be on the external surface of the head itself or the force of a collision occurring elsewhere on the surface of the body may be transmitted to the head by way of the spine.

Although the precise manner in which some of the mechanical disturbances of the brain are brought about is obscure, it has long been recognized that certain types of external force tend to cause more or less characteristic injuries. Thus, if the nature of the alleged accident or assault is known, it may be possible to predict the particular kind of structural or functional disturbance that is likely to have been sustained. Conversely, if the nature of the disturbance is known it may be possible to reconstruct the probable manner in which it was produced.

An injury of the head may involve the scalp, the skull, the meninges, or the brain itself. These structures may be involved singly or in any combination. Obviously, the principal significance of a head injury lies in the extent to which the brain is or will subsequently be affected. It may be damaged immediately by the disruptive mechanical force of the impact or it may be affected some time later because of damage done to the skull or to the meninges. It is important to bear in mind that severe and even fatal damage to the brain may be sustained without significant injury to the scalp or skull. Conversely, extensive damage to the scalp or skull may be sustained without concomitant injury to the brain. The inac-

curate characterization of an injury in which the brain has been damaged simply as a skull fracture is as regrettable as it is common.

INJURIES OF THE SCALP

The principal significance of a wound of the scalp in relation to the general problem of head injury lies in the fact that it provides objective evidence that force has been applied to the head. To the experienced observer it may indicate not only the direction in which the force was traveling but it may also disclose the character of the instrument or object that produced it.

Undoubtedly, the most susceptible large skin area of the body to disruption by blunt impact is the scalp. This is due to the fact that nowhere else in the body is such a broad expanse of skin separated from the underlying bone by so thin a cushion of soft tissue. In most situations the skin

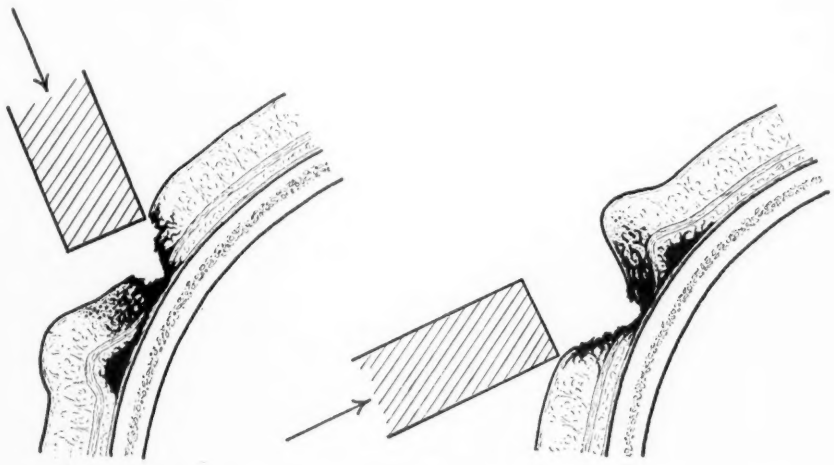

FIG. 1.—Lacerations of Scalp: Wounds of the type shown in A are commonly produced incident to assault when the head is struck with a blunt instrument from above. Wounds of the type shown in B are commonly the result of a fall in which the downward moving head meets resistance. (From Moritz, Pathology of Trauma, Courtesy of Lea & Febiger)

is well cushioned by fat or muscle so that an impact is likely to be decelerated slowly but this is true only to a very limited extent in the case of the scalp.

Because the skull is curved, the force of an impact is frequently deflected and the scalp is accordingly stretched and torn. Characteristically such lacerations tend to occur at right angles to the direction of the impact. Careful examination will usually disclose the direction of the impact by the manner in which the scalp has been torn from the skull. Thus in the case of a glancing blow from above the lower margin of the laceration is likely to be undermined and separated from the bone. In the case of an impact from below such as occurs when a person slips and falls to the ground, the upper margin of the laceration is likely to be undermined (Fig. 1). If the head is struck at an angle by a small object, a narrow tongue or three cornered flap of the scalp is likely to be torn loose. The

apex of the flap will mark the site of the initial impact. If the area of a glancing impact is large as much as half of the scalp may be torn from the skull.

Case 1.—The wife of a disoriented and agitated inmate of a mental hospital complained that her husband had been brutally assaulted by an attendant. A nurse had found her husband on the floor of his room in an unconscious state and bleeding from a deep wound of the scalp. The accused attendant stated that the patient was all right about two hours prior to the time that his injury was discovered, at which time he had brought the patient a bedpan. The wife stated that her husband had frequently complained that this attendant had threatened him with a club and that she had personally observed that the attendant was impatient and rough with his charges. She felt sure that the attendant had struck her husband on the head on the morning in question.

An examination of the scalp wound revealed that the force responsible for it had been traveling upward. An inspection of the premises disclosed that the injury had undoubtedly been sustained as a result of a fall in which the patient had struck the back of his head against the edge of a chair. This impression was strengthened by the fact that the injured man was known to be the subject of frequent fainting spells. Although the evidence upon which the wife based her charges was at no time strong, it was gratifying that the medical findings were sufficiently definitive to dispose of the case to the satisfaction of both the wife and the hospital authorities.

The nature of the object responsible for an injury of the scalp can frequently be recognized by the shape of the wound. Thus, the imprint left by a tire tread, by the grill of a radiator, by the head of a hammer, or by any one of a number of other objects may be clearly delineated by the pattern of the wound.

Case 2.—A maid left in charge of a town house was found dead on the floor by her employers when they returned from a week-end at the shore. She had been the victim of rape and had died as a result of head injury. Examination of the scalp and skull disclosed the fact that she had been struck repeatedly with an instrument having a triangular striking surface, the sides of which measured about one and one-quarter inches in length. The police investigation was directed to the finding of an instrument answering to this description. A blood-stained mason's hammer, the head of which corresponded to the size and shape of the wounds, was eventually found beneath the back porch of a gardener's cottage on the grounds. The similarity between the peculiar head of the hammer and the contour of the wounds was an important evidential factor in the subsequent conviction of the gardener.

The finding of a recent bruise or laceration of the scalp of a person unconscious from unknown cause does not justify the assumption that unconsciousness was caused by mechanical injury. People who collapse so unexpectedly that they fall to the ground frequently sustain secondary head injury. More times than not, the injury received in such circumstances is inconsequential, although it may appear otherwise at first. It is by no means uncommon in the case of a person found unconscious with a bruise or laceration of the scalp to find that the true cause of the collapse was heart disease or spontaneous intracranial hemorrhage.

Case 3.—A man arrested at midnight for drunkenness and disorderly conduct was found dead on the floor of his cell on the following morning, with evidence of

recent bleeding from the nose and mouth and with a large bruise of the forehead. He had shown no external evidence of injury at the time of his arrest. Relatives charged that the deceased had been assaulted by the police and their allegation apparently was corroborated by the preliminary findings of the coroner who reported that in his opinion death had resulted from a traumatic subarachnoid hemorrhage.

Subsequent investigation of the brain by a qualified pathologist disclosed that the fatal hemorrhage had originated from a miliary aneurysm of the circle of Willis, that the bleeding from this aneurysm had been in progress for several days prior to death,

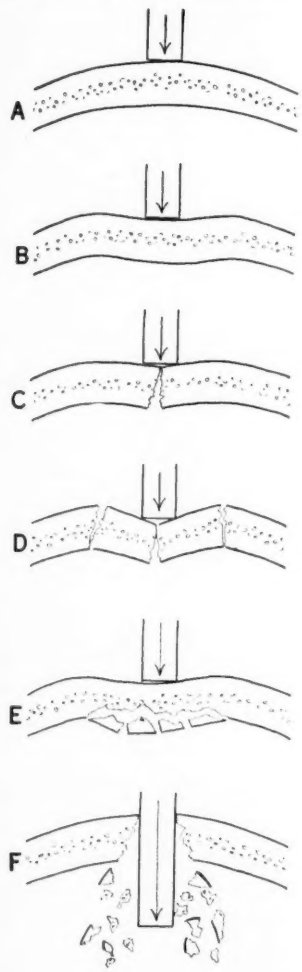

FIG. 2.

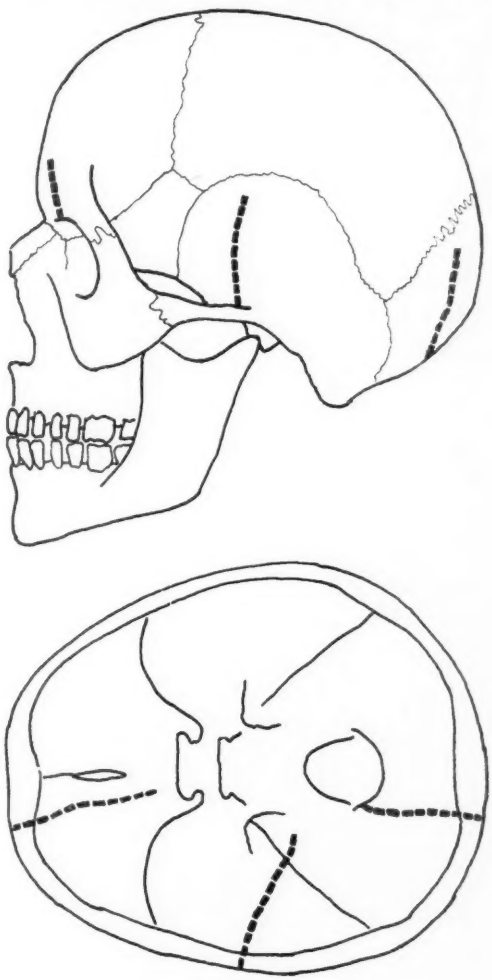

FIG. 3.

FIG. 2.—The Effects of Impact on the Skull: A. Before impact. B. Indentation or flattening, without fracture. C. Simple linear fracture. D. Comminuted fracture. E. Flaking of internal table (most commonly caused by glancing impact of bullet). F. Inverted crater (characteristic of penetrating bullet wound).

FIG. 3.—Schematic representation of characteristic sites of basilar fracture caused, respectively, by frontal, lateral, and occipital impacts.

and that mechanical injury had no causal relation to its occurrence. It was concluded, therefore, that the decedent had probably been disturbed during the night by headache caused by the accumulation of blood within the subarachnoid space, had arisen from his cot, fainted, and collapsed face down on the stone floor of his cell with resulting bruising of nose, lips, and forehead. The charges against the police

were withdrawn. The seriousness of the injustice that might have resulted if the true significance of the medical evidence had not been recognized is apparent.

INJURIES OF THE SKULL

Whether the bone breaks at the site of, or remote from, the external impact depends largely on the velocity at which the collision occurs and on the local resistance of the skull. Often the only objective evidence bearing on the manner in which a fatal head injury has been sustained is disclosed by the location and character of the lines of fracture. This fact is of particular importance to the coroner or medical examiner who finds in about 20 per cent of all cases of fatal head injury investigated by him that the postmortem findings are the sole source of objective evidence relating to the manner in which the injury was incurred. In such instances the only reliable evidence bearing on such important medicolegal questions as the number of times the head was struck, the site at which and the violence with which the force was applied, and the size and kind of object responsible for the injury may be disclosed by the autopsy.

In attempting to reconstruct the circumstances in which a given injury was sustained, three possibilities should be borne in mind. The destructive effects of an impact may be limited to the site to which the force was applied, may be situated at a distance from it, or may be in part local and in part distant.

FRACTURE AT THE SITE OF IMPACT

Bone is generally more susceptible to fracture by traction than by compression. Thus, since the initial effect of an external impact against the head is usually to compress the outer table and stretch the inner table the latter is the first to fracture. If sufficient indentation occurs a fracture which began in the inner table will extend through to the outer. Obviously the formation of a single crack is not the only form taken by the local injury. It is by no means unusual to find several lines of fracture radiating from a point beneath the site of impact. The presence of multiple systems of intersection fissures usually indicates that multiple impacts have been sustained. The momentary deformity created by an impact may be so great that concentric lines of fracture develop around the central point so as to bring the radiating fissures into communication with one another. Such a lesion tends to reproduce the web-like pattern often seen in a shattered pane of glass.

Case 4.—A man was found dead of head injury on a cement parapet at the bottom of a 12-foot embankment. The fact that the decedent was known to have been drunk when last seen alive, and that there was no guard rail on the embankment, led to the assumption that his death had resulted from an accidental fall. The disclosure at autopsy, however, that there were three distinct systems of fissures in his skull indicated that the injuries had resulted from assault rather than from accident. It was apparent that the dead man had been struck twice on the left side and once on the right side of the head with a long, heavy cylindrical instrument, which was estimated to measure about one inch in diameter. Further police investigation led to the apprehension of the assailant. An iron pipe had been used in the attack.

Occasionally the force of an impact is expended locally, so as to depress a circumscribed plate of the outer table into the diploe. If the vertical supports of the diploe beneath the site of the impact are sturdy, the external table may remain intact but portions of the inner table may be dislodged in the form of one or more flakes.

Bullet wounds of the skull bear certain highly characteristic features. The passage of a bullet through a flat bone ordinarily results in the production of a cone-shaped defect, the diameter of which is greater on the side of exit than on the side of entrance. Thus, large chips of the internal table may be dislodged and driven against the brain despite the

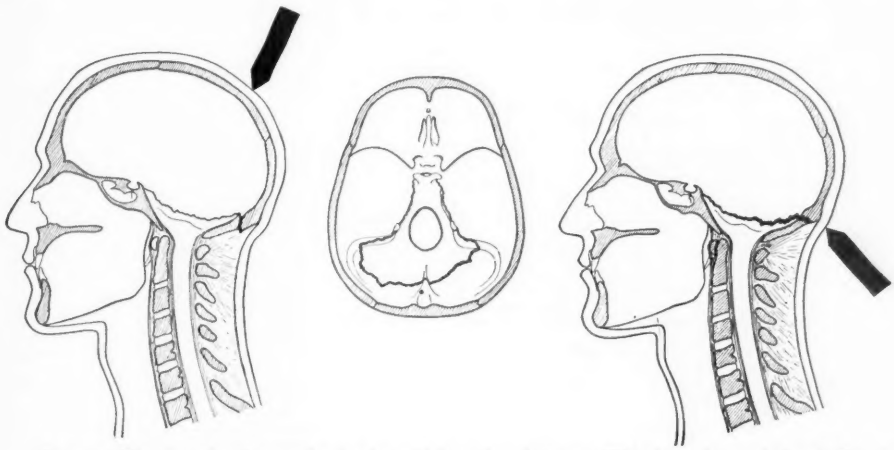

FIG. 4.—Diagram showing mechanics by which a ring fracture (B) of the base of the skull may be produced. In A the fracture resulted from an impact which tended to drive the base of the skull against the spine, and in C which tended to pull the skull away from the spine. (From Moritz, Pathology of Trauma, Courtesy of Lea & Febiger)

fact that the corresponding wound in the external table is small. Furthermore, the wound made by a bullet leaving the head is usually larger than the one produced where it entered. This is probably due to the fact that the bullet is often deformed or thrown off center by its first impact so that it presents a broader surface of impact when it strikes the bone on the opposite side. In the case of injuries by high velocity projectiles, fragmentation of a large part of the skull may result from the explosive effects of the collision.

Case 5.—Two small round defects were found in the skull of a badly charred body which was recovered from a burned dwelling. It was apparent to the pathologist that the defects had been produced by a bullet and that death had occurred prior to the conflagration. One of the holes was in the roof of the mouth and the other was in the top of the head. A gun, extensively damaged by the fire, was found near the body and it could not be determined whether it had been the property of the deceased or not. Obviously, the district attorney wished to know whether the death had resulted from suicide or homicide. The latter possibility was enhanced by the fact that the dead man was known to have kept a large sum of money on the premises.

The contour of the wounds in the bone provided clear evidence that the bullet had entered the skull by way of the month and that the muzzle of the gun was probably

in contact with the palate when the fatal shot was fired. It was concluded, therefore, that death probably resulted from suicide rather than homicide.

When the muzzle of the gun is held in contact with the head at the moment of firing, rapidly expanding gases of combustion may enter the cranial cavity with the bullet. In such an event the entrance wound in skin and bone may be large and it is not unusual to find that a large part of the floor of the skull has been blown out. Such explosive basilar fractures are likely to have a butterfly pattern and may or may not communicate with the wounds of entrance or exit. Occasionally, despite the fact that a bullet has glanced off the skull without penetrating it, fragments of the internal table may be detached and driven against the brain with sufficient violence to produce severe cerebral damage.

FRACTURE DUE TO TRANSMITTED STRESS

As already indicated the destructive energy of blunt force is frequently transmitted in such a manner as to result in a fracture at a considerable distance from the site of external impact. The two most important factors in determining whether or not disruptive stresses will be transmitted to other parts of the skull are first, the elastic properties of bone at the site of impact and, second, the velocity of the impact. If the skull is relatively inelastic at the site of primary impact it will resist deformity and break locally at the moment when the distorting force exceeds the cohesiveness of the bone. If the bone at the site of impact is elastic and if the energy of impact is liberated slowly enough to overcome its local inertia, the skull will be deformed and fracture will occur at the site of greatest mechanical disadvantage.

The base of the skull is comprised of relatively heavy bones which tend to break rather than bend when subjected to deforming force, whereas the bones comprising the vault are thin and relatively elastic. The base is accordingly more vulnerable to fracture when subjected to a deforming force than is the vault. Thus, a frontal impact characteristically results in a fracture which begins at or near the site of injury and runs backward through the base of the anterior fossa. An impact against the side or back of the head characteristically results in fractures which run, respectively, inward through the middle or anteriorly through the posterior fossa.

Fractures of the base, also, result from forces which tend to drive the skull against or to tear it away from the spine. Thus, a blow on the top of the head or a fall on the buttocks may force the skull against the spine with sufficient violence to break a circular or ovoid plate out of the floor of the skull around the foramen magnum. Similarly, the upward thrust of an impact from below against the back of the head may exert sufficient shearing stress at the craniospinal articulation to produce a circular fracture of the base. Such a fracture may encircle or communicate with the foramen

magnum. If the lateral limbs extend forward through the petrous portions of the temporal bones they are likely to communicate with each other in or behind the sella to complete the circular fracture anteriorly.

INJURIES OF THE INTRACRANIAL MEMBRANES AND THEIR VESSELS

A blunt impact to the head may injure the dura, the dura and leptomeninges or only the leptomeninges.

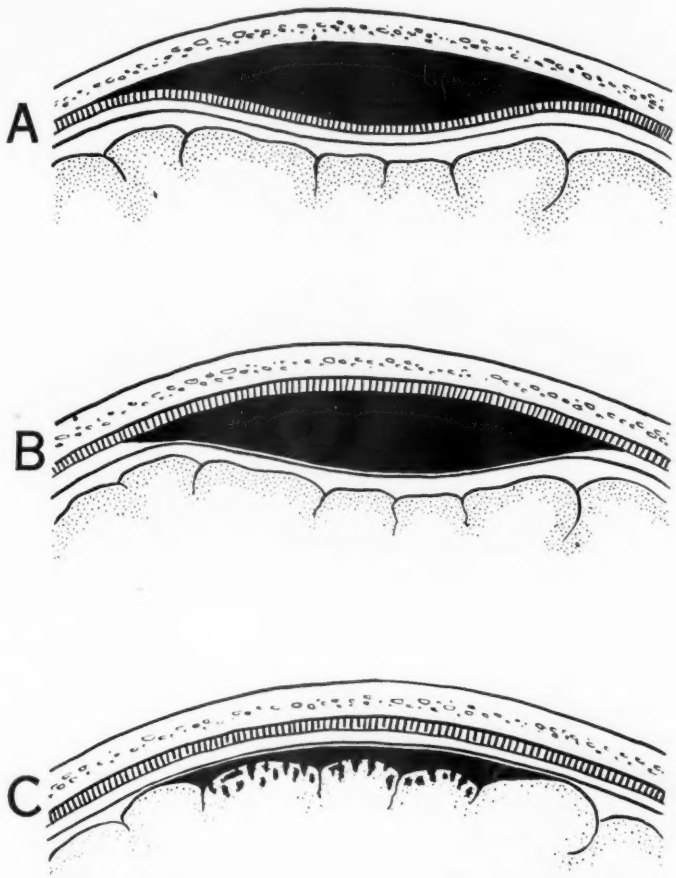

FIG. 5.—Common Sites of Posttraumatic Intracranial Hemorrhage: A. Epidural. Usually with fracture and beneath site of impact. B. Subdural. With or without fracture. May or may not occur at site of impact. C. Subarachnoid with superficial cerebral injury. With or without fracture. May occur at site of impact, at opposite pole of brain, or in both situations.

As already indicated, the dura is lacerated whenever the separation of the edges of the fractured bone exceeds the elasticity of that membrane. A defect in the dura is not in itself important except as it may create a portal of entry for infection, be associated with hemorrhage or predispose to the formation of adhesions between skull and brain. Dural defects in the vault or in the posterior fossa do not ordinarily predispose to infection. Defects

in the base, however, may lead to a direct communication between the pia-arachnoid and the middle ear, the accessory air sinuses or the nose.

Two sites of dural injury are of particular importance in relation to intracranial bleeding. One is in the vicinity of the great venous sinuses and the other along the course of the middle meningeal artery. Laceration of the former may lead to the rapid accumulation of a massive subdural hematoma and laceration of the latter often results in the formation of a rapidly fatal epi- or subdural hematoma.

It should be borne in mind that traumatic subdural bleeding frequently occurs without coexistent damage to the dura itself. A blunt impact, even though the skull remains intact, may cause sufficient agitation of the intracranial structures to lacerate the small veins that bridge the subdural space. Such vessels are most numerous in the vicinity of the sagittal sinus and beneath the under surface of the temporal lobes.

Case 6.—An elderly man, living alone in a rooming house, was found dead in bed one morning. He had been heard to enter his room at about 11:00 P.M. on the preceding evening and had appeared to be in his usual good health when last seen alive at 7:00 P.M. His door was locked from the inside, and there was a telephone on his bedside table. There was no external evidence of injury and it was assumed that the decedent had suffered a heart attack during the night. Since, however, there was no available history of previous heart trouble it was decided to perform an autopsy.

Postmortem examination disclosed a theretofore unsuspected bruise on the back of the head and a large recent subdural hematoma over the right hemisphere. Police investigation of the movements of the deceased during the evening prior to his death revealed that he had fallen while ascending the steps of a friend's house. According to the friend, he had been stunned a few moments but recovered sufficiently to play a game of cards. At about 10:00 P.M. he complained of a headache of increasing severity and had gone home.

It was apparent from the medical evidence that death had resulted from violence rather than from natural causes. The existence of a considerable amount of accident insurance made this finding a matter of practical rather than academic interest.

Old persons and chronic alcoholics are particularly susceptible to head injury, and subdural hemorrhage may follow impacts so mild that little or no immediate attention is paid to their occurrence. The bleeding is often so slow that days or even weeks elapse before its occurrence is recognized.

If, as is sometimes the case, the arachnoid is also torn, cerebrospinal fluid may escape into the subdural space and mix with the blood. Obviously, in such circumstances, there will also be an escape of blood into the cerebrospinal fluid.

The leptomeninges are likely to be damaged concomitantly with the dura if the latter is lacerated, and concomitantly with the cerebral cortex if the brain is severely bruised or lacerated. If both the dura and the arachnoid are lacerated at the same site cerebrospinal fluid is free to escape into the extracranial tissues or spaces. When such defects communicate with the nose, ears, or accessory air sinuses, leptomeningitis is likely to result.

If the arachnoid remains intact posttraumatic adhesions do not form between brain and dura. If, however, the arachnoid and dura are interrupted at the same site dense fibrous adhesions are likely to form. Such adhesions are strongly epileptogenic.

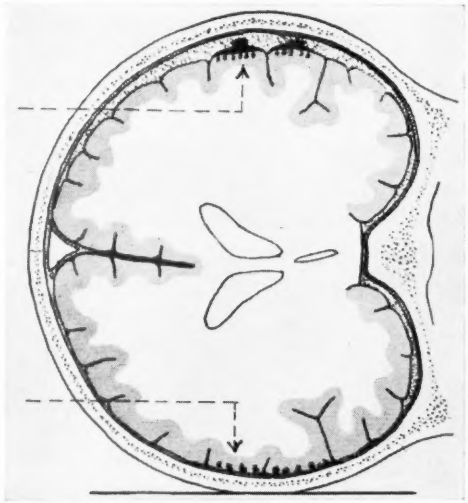

FIG. 6.—Impact of rapidly moving head against the pavement. Since the brain is decelerated less rapidly than the skull the cortex is likely to be bruised on the side of impact and lacerated on the opposite side.

CONCUSSION OF THE BRAIN

The phenomenon of being stunned or rendered temporarily unconscious by a blow on the head depends for its occurrence on a sudden change in the state of rest or motion of the head. If at the moment the head is struck it is supported in such a manner that it remains stationary, extensive injury to the brain may be sustained without loss of consciousness. However, if the motionless head is suddenly accelerated or if the rapidly moving head is suddenly decelerated concussion is likely to result. Recent experimental observations[1] have failed to disclose any characteristic morphologic changes incident to the receipt of this type of injury. Contrary to opinions held in the past, the sudden rise in intracranial pressure caused by external impact does not appear to be responsible for the phenomenon, nor does it appear to depend on the occurrence of cerebral ischemia due to reflex vascular spasm. Obviously, the occurrence of a concussive injury does not exclude the possibility of other forms of cerebral damage. By the same token, posttraumatic unconsciousness is not invariably the result of concussion. Although a fall to the pavement may result in such rapid deceleration of the moving head as to cause loss of consciousness, the same impact may result in cerebral damage so severe as to account for unconsciousness independently of concussion. Even though concussion is not sustained the progressive elevation of intracranial pressure due to hemorrhage from damaged

blood vessels may cause unconsciousness. The latent period between injury and loss of consciousness from hemorrhage may be seconds, minutes, or many hours, depending upon the rapidity of the bleeding. An individual who is "knocked out" by a fall and who regains consciousness after a few minutes, only to lose it again after a few hours because of intracranial bleeding, constitutes a good example of both "concussive" and "nonconcussive" unconsciousness.

CEREBRAL INJURIES BY BLUNT VIOLENCE

The impact of skull against brain beneath the site to which external force is applied may result in contusion, laceration, or crushing of the underlying cortex. The force may be sufficient to destroy a large part of a lobe, or it may be expended in the production of a shallow bruise of the eminences of one or two of the subjacent convolutions. After either bruising or laceration localized ischemia induced by the interruption of small blood vessels may result in secondary enlargement of a lesion. The contraction of scar tissue at the site of a cerebral injury may result in the development of functional disturbances from a theretofore silent lesion.

Neither contusion nor laceration is necessarily limited to, nor do they necessarily occur in continuity with the site of external impact. If the impact is sufficiently severe the brain may be displaced so as to collide with a fold in the dura with sufficient violence to cause laceration. Thus, the corpus callosum may be torn by being thrust against the falx cerebri or the peduncles may be lacerated by collision with the tentorium. When such lesions are produced, other more severe injuries to the brain are usually sustained.

The mechanism by which *contrecoup* injuries of the cortex are produced is not always clear. The impact of a moving head against a stationary object frequently results in cortical damage on the side opposite that which sustained the external impact. Thus, a fall on the back of the head frequently results in the production of more extensive cortical injury to the frontal than it does to the occipital lobes. Similarly, it is not unusual for a blow over the left hemisphere to result in damage to the right or for a blow over the frontal region to cause injury of the occipital lobes. Occasionally contralateral injury results from an impact which neither sets the head in motion nor brings the moving head to a stop. In such an event the contralateral lesion is most plausibly explained on a basis of contusion. The skull is compressed bilaterally and the brain is squeezed between the inwardly moving sides.

In most instances contusion fails to provide a satisfactory explanation for *contrecoup* injury because the nature of the trauma is such that the occurrence of a contralateral impact between skull and brain is extremely unlikely. The most common type of injury to result in a *contrecoup* lesion of the cortex is a fall in which the rapidly moving head strikes a resistant object. In such an event, the forward momentum of the brain will tend

to tear it away from the side of the skull opposite the point of impact, and the traction thus exerted on the arachnoidal trabeculae leads to laceration of the underlying brain substance.

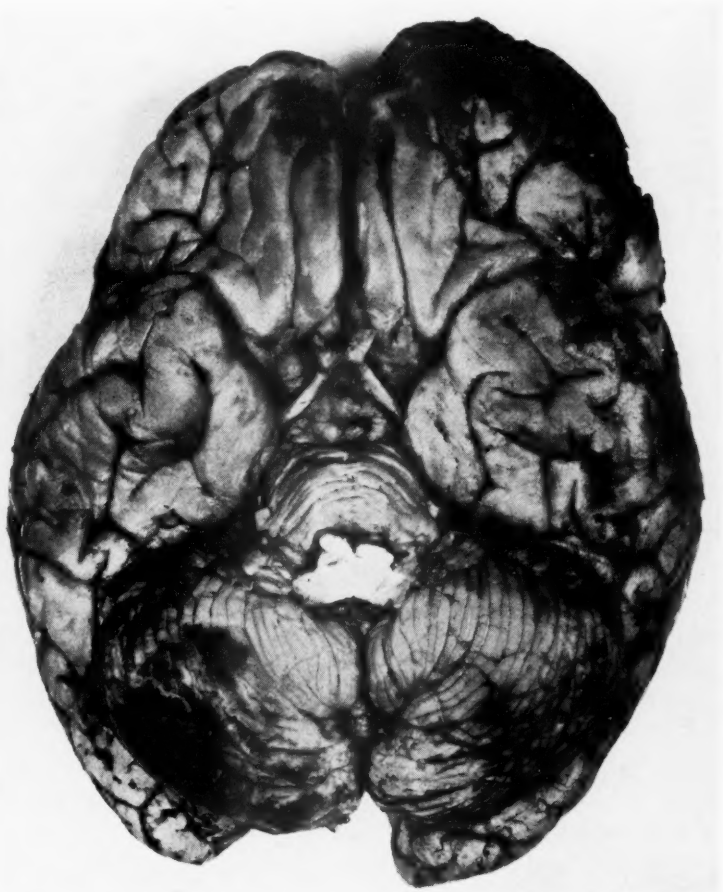

FIG. 7.—Photograph of the under surface of a brain showing the effect of direct and *contrecoup* injury. The external impact was sustained on the right side of the back of the head. The direct effect of the blow is seen on the surface of the right lobe of the cerebellum and the *contrecoup* injuries are most pronounced at the anterior pole of the left frontal lobe.

DIFFUSE CEREBRAL INJURY BY BLUNT IMPACT

The mechanism by which blunt impact produces diffuse in contrast to local injuries of the brain is often obscure. Whereas superficial bruising and laceration can be attributed to compression or traction, no such simple explanation suffices to account for the widely disseminated internal lesions that are occasionally sustained. Such lesions usually take the form of multiple petechiae or scattered foci of degeneration and necrosis. That they are probably caused by agitation of the brain as a whole is indicated by the fact that they are sometimes observed independently of a recognizable cortical

lesion. Disseminated foci of progressive gliosis following nonpenetrating injuries of the head probably have their origin in such lesions.

PENETRATING INJURIES

There is little to be said regarding the mechanism of injury incident to penetration of the brain other than to call attention to some of the special characteristics of wounds produced by bullets. Regardless of the size, shape or velocity of a bullet, when it strikes the skull with sufficient force to penetrate, it usually produces a larger defect where it emerges from the inner table than where it enters the external table. In doing so multiple fragments of bone, each constituting a secondary missile, may be driven into the cortex in the vicinity of the tract of the bullet.

FIG. 8.—Photograph showing the forward and lateral thrust imparted by a bullet in its passage through clay. It is to be noted that the diameter of the resulting defect is much greater than that of the bullet. The muzzle of the gun was approximately 18 inches from the target at the moment of firing.

Frequently the bullet is thrown off center as a result of its impact against bone so that its wobbling produces a larger defect in the brain than would be expected from the diameter of the projectile. Whether it wobbles or not a bullet imparts a lateral and forward thrust to the medium through which it passes, the result of which is the production of a broad cylindrical- or spindle-shaped defect in a tissue as soft as the brain. High velocity bullets such as are fired from military weapons and from certain sporting rifles are likely to disintegrate into many fragments as they pass through the body. Each such fragment becomes a secondary missile and contributes to the total effect of the injury.

SUMMARY AND CONCLUSIONS

It has been attempted in the foregoing discussion to relate some of the more common traumatic lesions of the scalp, skull, meninges, and brain to the kinds of mechanical disturbance responsible for their production.

From a medicolegal standpoint, it is important not only to know the kind of lesion that may be regarded as the natural consequence of a given type of injury, but also to be able to reconstruct the probable circumstances of an unwitnessed injury from the character and location of the anatomic lesions.

Mechanical injuries of the head have been classified as penetrating and nonpenetrating. The principal differences between penetrating and non-

penetrating head injuries lie in the fact that the former are more frequently associated with the presence of intracranial infection, ordinarily involve greater primary destruction of brain tissue, and are more frequently followed by the formation of craniocerebral and intracerebral scars.

The immediate loss of consciousness following injury of the head may be due either to a sudden agitation of the head, with or without the production of recognizable morphologic changes, or to direct mechanical disruption of brain tissue independently of agitation of the head as a whole. Delayed onset of unconsciousness or nonrecovery from immediate posttraumatic unconsciousness usually indicates the occurrence of intracranial bleeding, with an associated increase in intracranial pressure. Such bleeding may be epidural, subdural, subarachnoid, or intracerebral. Other sequelae of mechanical injury which may be responsible for delay in the onset or prolongation of the duration of unconsciousness include secondary cerebral vascular disturbances, edema, and infection.

Residual posttraumatic disability after apparent recovery from a head injury may be due to the contraction of craniocerebral adhesions or intracerebral scars, to secondary cerebral circulatory disturbances, to the presence of intracranial foreign bodies, to the presence of intracranial infection, or to obscure causes not associated with recognizable gross or microscopic abnormality.

REFERENCE

1 Denny-Brown, D., and Ritchie Russel, W.: Experimental Cerebral Concussion. Brain, **64**, 93, 1941.

FORENSIC ASPECTS OF BURNS

SPECIAL REFERENCE TO APPRAISAL OF TERMINAL DISABILITY

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THE BURNED PATIENT presents a great many problems that concern the lawyer as well as the doctor. The immediate problem of saving life and restoring the burned individual to society is the primary care of the physician, but, unfortunately, the final outcome frequently is complicated by factors producing results that are far from perfect. These factors are of interest to the lawyer who is attempting to determine the proper methods for evaluating legal settlements. It is true that the lawyer can turn to the medical profession for aid, but he himself should know something of the fundamentals presented by the burned patient to enable him to more quickly, and more justly, complete his work.

In peace-time burns occur all too frequently. According to statistics published by the National Safety Council in 1940 and 1941, burns killed 23 per cent of all children under the age of five who died from accidental means. From age 5–15, burns killed 13 per cent of all those dying violent deaths, and from 15 years on burns took a yearly toll of six per cent of all victims exterminated by accidents. There are no statistics to indicate the number of people who received burns but lived. However, from the figures obtained at the Boston City Hospital, where there is a very large burn population, it is evident that about 1000 patients receive burns and live, for the one who is burned and dies. Approximately ten per cent of those that live are badly enough burned to be hospitalized. It is in this group that the many problems of interest to the lawyer occur.

There are a number of angles which have to be studied by the lawyer to arrive at his final decision. Most of these can be estimated if he knows the cost of medical care, the time-element involved, the immediate disability after healing is complete, and the probabilities and possibilities regarding future pathology.

DEFINITION OF A BURN

A burn is a loss of continuity of the body surface due to a coagulation and destruction of skin and subcutaneous tissues by thermal changes, including both heat and cold, by chemicals, by electricity, and by radiation. No distinction should be made between a burn and a scald, inasmuch as they are pathologically the same, and must be considered identical from the viewpoint of physiology and treatment.

CLASSIFICATION

The classification of burns refers to the depth of the pathology or to the amount of tissue involved. In the past, the classification of Dupuytren was

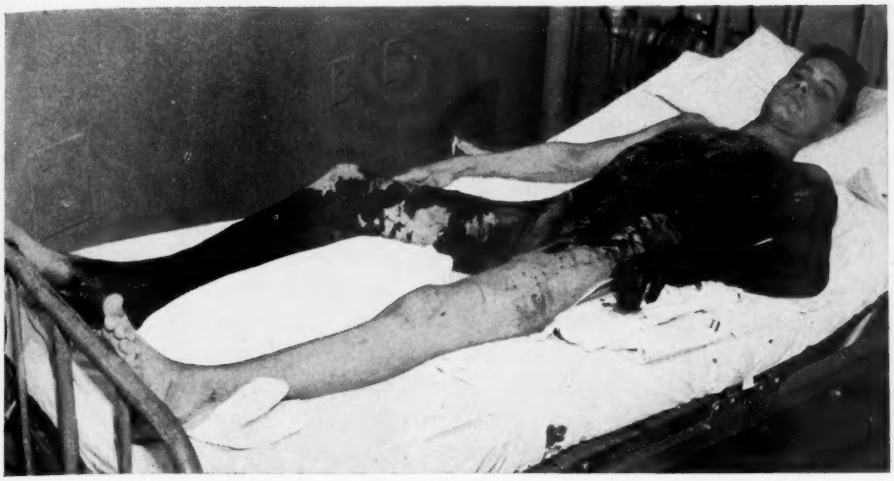

FIG. 1.—This patient received a severe electrical burn from a power line containing 33,000 volts. The current entered the left hand, which was upraised, entering the palm and fingers as a spark. The spark ionized the air, thereby producing conductivity to the rest of the arm. Most of the power kept sparking-back into the cable from the left arm, but enough current remained, which passed across the chest and abdomen and down the right leg, to ground itself on a steel girder beneath the right foot. The heat was so intense that it set the clothing on fire. The patient was immediately knocked to the ground and did not breathe for three hours and ten minutes. He was kept alive by artificial respiration. After treatment for shock he received the aniline dye treatment on the burns. His convalescence was quite stormy due to many complications. He had an amnesia for two weeks after the burn took place. There was injury to the left kidney due both to infection and to the electrical current passing through the kidney area. The left arm was almost completely cooked, and at many times it was considered advisable to remove the arm. However, as this procedure is irreversible, the arm was left on. It can always be removed in the future if it gives him further trouble. The patient had special nurses for four months, and had six months of hospitalization. He had repeated transfusions and skin grafting was necessary on the left arm. The final result was excellent both from the cosmetic and functional points of view except for the left arm.

In the final award on the case the sum paid was based on the expense of the medical care and the suffering the patient underwent. The factors of later terminal disabilities were taken into consideration. The possibility of cancer was considered, and it was also brought out that the left kidney was probably partially damaged, and that sterility would probably result. The two weeks of amnesia following the burn indicated brain damage, and this was also an element in the verdict.

The patient in this case brought suit against both the power house where he was making a survey and the technical school that he was attending. Electrical experts established the fact that the power box on the wall, which was the source of the current that injured the boy, was not marked as being dangerous and was placed too close to the floor.

Medical testimony was introduced to show the extent of the injury and the probabilities and possibilities of future disabilities. The judge gave a verdict against the power house of $15,000. $5,000 was paid for hospitalization, nursing and medical care. The actual bills amounted to this sum. The additional $10,000 was allowed to compensate for the suffering the patient went through, and to allow him to complete his education for rehabilitation in society. It was recognized that the left arm was useless, and that future complications may prevent the patient from earning his own living for some years.

This award is still held in escrow until the case has been brought against the technical school.

advocated. This listed seven degrees, and was quite confusing. A much simpler form of classification has been adopted in which the burns are divided into three degrees. A first degree burn is one in which there is simply an erythema of the area involved. An example of this is the common sunburn. A second degree burn indicates a destruction of the epidermis. This is roughly the outer half of the skin. In this type of burn there is usually blister formation due to the exudation of plasma from the deeper layer of the epithelium which elevates the epidermis. A third degree burn indicates involvement of the full-thickness of the skin and any of the subcutaneous tissues. When the deeper structures are involved there is no change in the classification, the burn still being referred to as a third degree one.

THE PATHOLOGY

When a burn is produced by thermal changes and chemicals there is immediate destruction of the tissues involved in the local area. The destruction is one of coagulation, the living cells are converted into necrotic tissue, the blood and nerve supply to them are interrupted, and they become a foreign body to the host. Shortly after this occurs there is a dilatation of the capillary blood vessels around the burned area. Those capillaries near the site of the burn undergo a change in the permeability and allow plasma to escape into the interstitial spaces. This escape of plasma compresses the return flow of lymph. Outside of the area of local edema there forms the so-called pyogenic membrane, which consists of a thin wall of round cells. These leukocytes act as a defense mechanism to prevent invasion of the body by germs. The edema of the burned part usually reaches its peak in 12–18 hours, and remains for a period of three to seven days. At the end of that time there is reabsorption of the lymph and repair processes begin to set in. The exact nature of these depends entirely upon the site of the burn, its size, and the general condition of the patient.

Burns of roentgenray origin and electrical burns are apt to be slower in manifestation and much more extensive due to the penetration of the electrical energy. The destruction of tissue, however, is just as concrete and involves the body in the problem of repair.

SHOCK

Burn shock is a manifestation of symptoms and physical signs seen in practically all large burns. The mechanism of shock is still not completely understood. It is known that in shock, whatever may be its origin, there is a certain specific syndrome. This consists of a fall in blood pressure, associated with a decreased coronary output, a change in the capillary permeability on the body as a whole, releasing plasma from the blood stream into interstitial spaces, and a general depletion of all of the vital functions due to anoxemia, lowered venous pressure and a depletion of the circulation.

In most major burns the factor of shock is the immediate problem, and must be combated with heat, rest, the control of pain, and intravenous replacement therapy if the patient is to survive. The amount of shock depends on the size of the burn, the part of the body burned and, to a certain extent, with the speed in which the burn takes place. It is conservative to state that at least 75 per cent of the deaths that occur in the first 72 hours following the inception of the burn are due to shock.

INFLUENCE OF SIZE OF BURN ON DISABILITY AND LIFE

Burns can be divided into the classification of major and minor, depending on the surface area involved: Empirically, a minor burn is one in which less than 15 per cent of the body surface is destroyed. A major burn is one in which there is involvement of more than 15 per cent. This may seem, at first, to be a didactic classification, but it is essential when it is realized

that minor burns usually need only local treatment with no thought of shock, whereas the major burns invariably need shock therapy as well as treatment to the local areas. There is a limit to the amount of body surface that can be burned without producing death. Until recently a burn of more than one-third of the body was considered fatal. At the present, because of advancement made in local treatment and the use of intravenous plasma, it is now possible to save burns that involve up to 65 to 70 per cent of the body area. It is obvious that the larger the burn, the more profound the shock, and that the incidence of disabilities increase in direct proportion to the area involved.

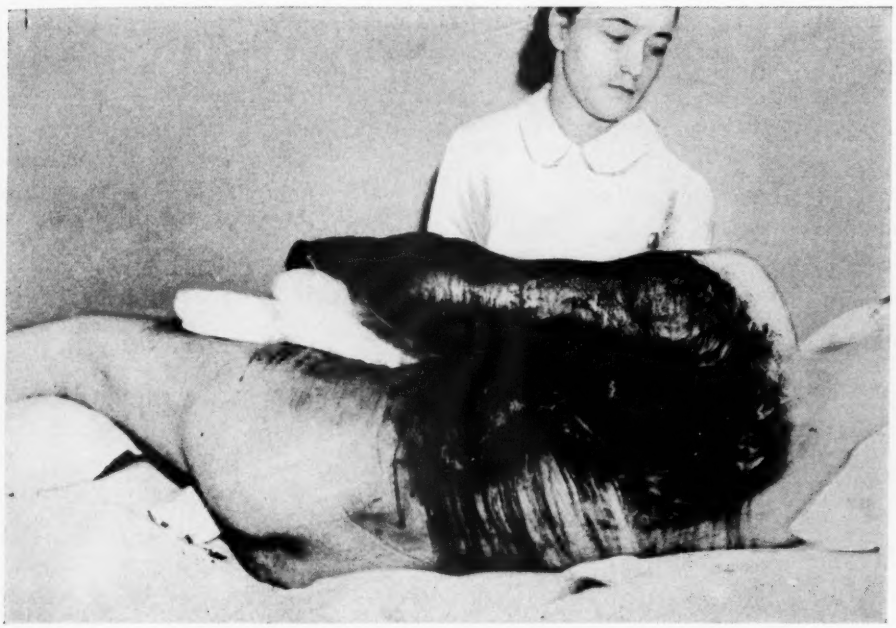

FIG. 2.—Back view of same patient showing the extent of burn on the posterior aspect of the left shoulder and the back of the left side.

INFLUENCE OF AGE ON DISABILITY AND LIFE

It is still debatable as to what factor age plays in the recovery of the burned patient and in his disability if he does recover. In general, it can be stated that very young children have little reserve strength to combat their pathology. Older children and young adults can withstand a tremendous amount of trauma and apparently have much better powers of healing. Corrective measures such as plastic surgery are more successful in children than in adults.

RESTORATION AND HEALING OF THE THREE DEGREES OF BURNS

In order to understand the complications and disabilities that may result from a burn, it is essential to understand some of the fundamentals of the repair processes. Nature attempts to restore the body to its original pattern,

but the end-result is not always ideal. The factor of the depth of the burn has a large bearing on what the final outcome will be and the disabilities that may result.

A first degree burn heals by a return of the erythema to the normal circulation and by desquamation. The hornified layer of the epithelium peels or flakes away over a period of a few days. The underlying skin builds up a new hornified layer. This is done with no scarring and with no real disability except that caused by pain or by the systemic reaction which is usually present for only 24 to 48 hours. The systemic reaction probably results from both pain and the action of the actinic rays on the body. There can be no absorption of a poisonous protein, and there can be no infection as there is no portal of entry caused by a break in the skin. In severe sunburns, heat prostration or sunstroke may occur.

Second degree burns heal by building up an intact skin from the dermis. The epithelial cells composing the dermis have to undergo a change in character to assume the rôle of the epidermis. The fact that the floor of the burn is paved with epithelium results in fairly rapid healing with little, if any, permanent disability or deformity. Second degree burns are apt to be more painful than the third degree type because the nerve endings in the skin have not been killed, but have been exposed. This increase in pain is apt to produce a greater degree of shock. The dermis has little immunity to infection, and may allow a severe infectious toxemia to occur. If second degree burns are cared for properly and the dermis is not killed by infection or by harsh local treatments, there is never need for skin grafting, and the cosmetic and functional result will be almost perfect.

Third degree burns heal in one of two ways, depending upon the depth of the burn. If only the full-thickness of the skin is destroyed healing will eventually occur by the spread of the islands of epithelium that are beneath the skin at the bases of the hair follicles and sweat glands. These islands will eventually come to the surface of the granulating wound and will spread and coalesce, giving a scar that is quite thin and flexible. This scar, while it is not always ideal in appearance, usually does not disturb the function of the part involved. When the burn involves the entire skin and the underlying tissues, healing can only progress from the edges of the wound or from skin transplanted from other parts of the body. This is the type of burn that gives rise to severe contractures and deformities if too long a time-element elapses before the burned surface is covered. In the granulation tissue that builds up in the burned areas contracture bands are formed from fibroblastic cells. These contracture bands can be very dense and can produce many untoward results.

IMMEDIATE DISABILITY FOLLOWING A BURN

The immediate disability following a burn consists of shock and toxemia. The shock occurs very shortly after the burn takes place and may last from only an hour or two up to a week or ten days, depending on the size of the burn and the treatment instituted. The toxic phase, due to infection, begins

about 40 hours after the burn takes place and may persist until healing is almost complete. In severe burns this may be for four or five months, or even longer. There is also the emotional factor to be considered. It is usually the active healthy child or the well individual who gets burned. The rapid transition from being perfectly well to a bedridden patient reacts strongly on a large percentage of the stricken individuals. They are usually frightened, apprehensive of death, and they worry a great deal about their being restored as normal individuals. The adult male is prone to wonder about his ability to support himself and others, and the female is inclined to become very depressed regarding her final appearance.

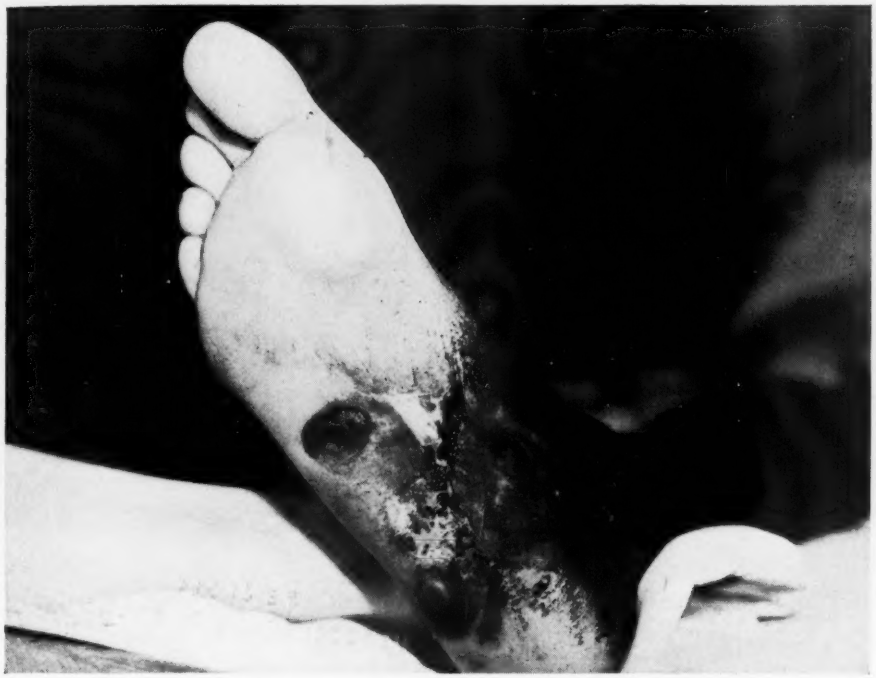

FIG. 3.—View of foot showing where the current arced from the foot into a steel girder.

IMMEDIATE DISABILITY FOLLOWING THE HEALING OF THE BURN

The disability following the healing of the burn is due to a number of factors. The more common ones are disabilities due to scar formation, to the development of keloid, to contractures in general, to lowered resistance to other diseases, to psychic trauma, to the development of unwanted emotional complexes, to inability to assume former rôles in society, and the loss of strength and activity. These factors are all perfectly obvious and self-explanatory and need not to be gone into fully. All of these factors, however, are very definite and must be considered in trying to evaluate the terminal disability of the burned patient.

LATE DISABILITIES FOLLOWING HEALING

Some of the disabilities listed above may put in their appearance many weeks or months after the burn has apparently healed. It is possible for

contractures and keloid formation to occur in burns that are fairly old. If such contractures interfere with the movements of the body, it is impossible for the individual to assume all of his former functions and duties. Frequently, contractures can occur around orifices of the body preventing or interfering with the natural functions such as defecation, urination, sexual intercourse, and even eating. Late disabilities are prone to produce a lack of social adjustment, thus having a direct bearing on the emotional stability of the patient.

COMPLICATIONS AND MORBIDITY

When a patient has undergone a long period of convalescence characterized by a profound state of shock in the beginning and many months of infectious toxemia, many organs of the body become injured beyond repair even though they are far removed from the site of the burn. Renal complications, such as nephritis, may have a permanent influence on the general health of the patient and his life expectancy. Prolonged infection can also injure the heart and may force the patient into a sedentary life. Electrical burns may cause permanent nerve or brain injury and may have a bearing on the sexual development and the sex life of the patient if the current involves the gonads. It is also a well-established fact that skin cancer is much more prone to develop in the scars produced by burns than in normal skin. All of these complications must be considered in evaluating the possible or the probable end-result the patient may expect.

THE COST OF BURNS

Because burns result in such profound and immediate disability, they are apt to be exceedingly costly to the individual or to the institution caring for the individual. All patients with large burns need very special care and constant attention. From the medical aspect large burns must be seen by the physician at least once a day and in extreme cases three and four times a day. These burns need trained nurses in constant attendance. Frequently, consultations with specialists must be obtained to determine plans of treatment. The treatment itself consisting, as it does, of large quantities of plasma, special diets and expensive vitamins, is not a cheap one. After the patient is healed he may have to undergo extensive plastic operations, involving more hospitalization and lost time from productive work. Burns of one-fourth of the body area, or more, produce absenteeism from employment for weeks and months. This loss of production has to be counted in as a part of the cost produced by the burn.

ESTIMATION OF THE DISABILITY AS A WHOLE

The estimation of the final terminal disability is a composite picture. The lawyer or the doctor, or both, must take into consideration all of the factors that were present from the time the burn took place, and sometimes it is necessary to go back many years before that. In general, it is considered

that there are ten chief factors or fundamentals that have to be considered. They are:

(1) Degree of the burn.
(2) Size of the burn.
(3) Part of the body involved.

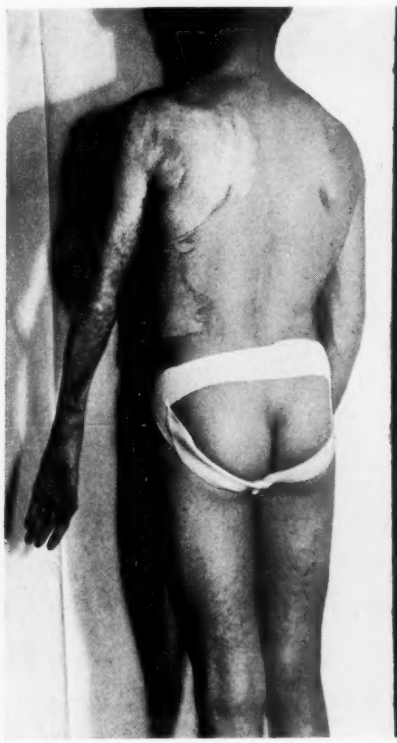

FIG. 4.—Posterior view following healing.

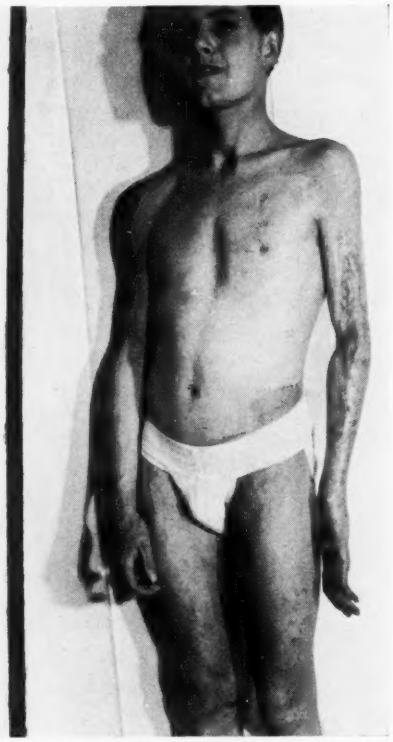

FIG. 5.—Front view of patient after healing. Note the minimum scarring of abdomen, chest and right leg. The left arm is withered and is almost a total disability.

(4) Age.
(5) Physical condition of the patient before the burn took place.
(6) Sex of the patient.
(7) Amount of terminal disability produced by the burn.
(8) Amount of psychic trauma produced by the burn.
(9) Occupation of the patient.
(10) The probabilities and possibilities of future morbidity.

These factors are quite obvious. It would require too much space to go into a complete discussion of each one of the above ten headings. It should be obvious, however, that, in most burns, the majority of the ten factors, if not all, will come into play, and the final evaluation of disability and the proper settlement to be made must be based on some such schedule.

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MINIMAL CRITERIA REQUIRED TO PROVE CAUSATION OF TRAUMATIC OR OCCUPATIONAL NEOPLASMS

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THE TERM TUMOR as generally used indicates a new growth of cells which is independent of the normal restraint exerted by the body on its tissues, but may also be used very broadly to indicate any swelling regardless of its nature. However, in the medical sense, usually a new growth or neoplasm is meant. In general, tumors are classified according to the type of cell which they tend to reproduce.

The diagnosis of a tumor should not be accepted without incontrovertible proof of its nature, obtained either by pathologic examination of the entire specimen when removed, or by biopsy. So many possibilities for error exist in the diagnosis of a tumor by clinical means, even when supported by radiologic evidence, that pathologic proof should be insisted upon in addition. Not only is accurate diagnosis and classification of a tumor impossible without microscopic examination of a section obtained from the substance of the tumor, but also microscopic examination gives important information as to probable rate of growth.

Since the causation of tumors is not yet clearly understood, it is inevitable that a great mass of speculation should have been built up on this subject. In general, it can be said that there is no one cause for cancer, any more than there is one cause for inflammatory diseases, but rather there are a number of causes, some known, some unknown, which produce different types of tumor. Some tumors are clearly congenital, others are related to endocrine disturbances; still others are due to the action of specific chemical or physical agents on the body tissues; still others are of unknown origin. We have learned from observation of cases in human beings and from experiments in animals that the response to a given agent acting in a given degree is not necessarily uniform, that there are individual variations, that even in the case of highly purified hydrocarbons which have been shown to produce cancer in experimental animals, a dose that will produce a cancer in one animal may not produce it in another.[1]

While trauma, in the broad sense, is any damage to the body, as ordinarily meant medically it implies the application of mechanical force to the body with sufficient violence to produce a break in the continuity of one or more of the body tissues. Repeated minor traumas may be spoken of as chronic irritation. As ordinarily used, the term traumatic cancer implies a malignant tumor which has arisen following a single mechanical injury. While there is much legal and scientific evidence that certain types of substances acting

upon the body over a period of time may produce certain types of benign or malignant tumor, so far as a single mechanical injury producing trauma is concerned, the evidence rests chiefly on reasoning from *"post hoc ergo propter hoc"*—all too often fallacious.

SECTION I—TUMORS DUE TO MECHANICAL TRAUMA

The minimal criteria necessary are: First, the integrity of the tumor site prior to injury must be established; second, the injury must be sufficiently severe to disrupt the continuity of the tissue at the site, and so initiate reparative proliferation of cells; third, the tumor must follow the injury by a reasonable length of time; and fourth, the tumor must be of a type which might reasonably develop as a result of the regeneration and repair of those tissues which had received the injury.

These criteria have been evolved gradually,[2, 3, 4, 5] and appear sound in the light of our present knowledge.

The mere coincidence of two rare events, such as an injury in an unusual part and the subsequent development of a tumor in that part does not necessarily establish a causal relationship.

One requisite, sometimes mentioned in the literature, for the implication of a single trauma as a causative factor in tumor production is the presence of so-called bridging symptoms; that is, symptoms that continue to give evidence of the continuance of disability from the time the injury is sustained to the time the tumor makes its appearance. Among these are continuity of pain, persistence of swelling, persistence of induration or of ulceration. However, this group of symptoms is of little practical importance in establishing causal relationship with the trauma and has no bearing either from the negative or positive standpoint. Even in instances where a tumor is incited by the subcutaneous injection of carcinogenic substances in experimental animals, such as methylcholanthrene, there may be no continuity of signs from the time of injection to the time of the appearance of the induced sarcoma. On the other hand, merely because inflammation, ulceration or swelling have been present in a region, there is no certainty that the subsequently diagnosed tumor is in any way due to the conditions associated with the persisting signs and symptoms. Indeed, such signs and symptoms may actually be due to a tumor already present before the injury and masked for a time by the inflammatory and reparative processes following the trauma.

The previous integrity of the part may be assumed if its appearance and function have been normal. Only rarely is a thorough medical or radiologic examination available for reference in this regard. In many instances local trauma only calls attention to a tumor already present but unnoticed.

The adequacy of trauma is usually not difficult to determine. The injured person usually has had a careful medical examination soon after the event and there is often observation of the injured part at frequent intervals for an extended period of time. These observations may be made by the simple means of inspection and palpation or they may be supplemented by various

types of radiologic and laboratory examinations. It may be fairly stated that trauma which fails to produce either an extravasation of blood into the tissues or a break in continuity of some of the tissues in the part affected is inadequate to be considered capable of initiating a tumor.

The time interval that may intervene between a trauma and the development of the tumor at the site is very difficult to determine. Any evidence

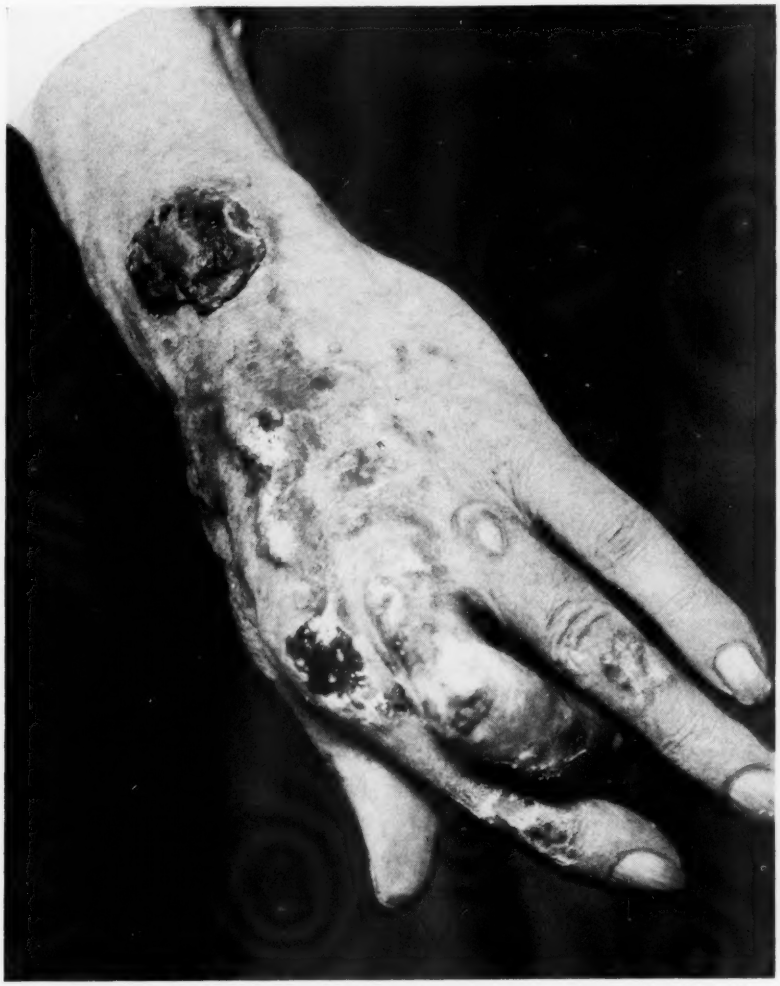

FIG. 1.—Multiple keratoses and epidermoid cancer due to prolonged occupational exposure to the roentgen ray.

bearing on the rate of growth of the tumor, either from clinical observation or pathologic study, is most helpful. In general, four weeks may be regarded as the minimal time for the appearance of a rapidly growing tumor following the receipt of an adequate injury. A maximal limit is difficult to set, but probably should not be over three years after the injury.

The type of tumor developing at the site of injury is important. Obviously, any dermoid or teratoid tumor, other than in the testicle, would be

automatically ruled out. Any tumor of cell type foreign to the part would be considered metastatic rather than primary.

Curiously enough, although sarcomas of bone are among the tumors most often considered as of traumatic origin, they practically never appear after the thousands of fractures that annually provide adequate trauma for tumor development.

Knox[5] stated, in 1929: "Much of what has been published (in regard to traumatic cancer) is utterly unprofitable to read"—unfortunately, this holds as well today.

Shear[6] notes that the application of mechanical trauma to the point of injection of methylcholanthrene exerts no accelerating effect on tumor formation and, possibly, inhibits the development of the tumor somewhat.

Much consideration has been given to tumors developing in scar tissue, thus indirectly relating the genesis of tumors to a previous trauma. Traumatic epithelial cysts are known to occur as a result of trauma driving epithelium down into the deeper tissues, but have little clinical significance.[7] It has been clearly established that the epithelium overlying the scars of burns is peculiarly likely to become malignant. If there is a chronic persisting ulcer in the area of the scar, this is even more likely to bring about development of malignancy. Ullman[8] states that as high as 80 per cent of these cancers developing in scars occur in the scars of burns.

Fischer-Wasels[9] felt that only those scars following freezing or burning might be a source of carcinomatous change. This concept is too narrow, however.

The latent period before scars become cancerous may be extremely long, instances of 50 years or more having been reported.

Certain types of cancer may develop following chronic irritation,[10] where repeated trauma activates degenerative and attempted reparative changes over a long period of time. While the great majority of chronic ulcers never become malignant, in a few, the attempted reparative processes may ultimately lead to neoplastic proliferation. In instances where this type of origin of tumor is alleged, the contributory negligence of the employee in permitting the chronic irritation to continue without adequate medical treatment should be carefully weighed.

One of the most difficult points to decide is whether or not trauma can change a benign tumor into a malignant one or can increase the malignancy of a cancer already present. It is, of course, obvious that if the malignant tumor is of a diffierent cell type from the preexisting benign tumor, there can be no relationship. On the other hand, if it is a malignant variant of the same cell type the question remains open. Here, no general rule can be laid down, but in coming to a decision, it must be remembered that benign tumors may take on malignant characteristics entirely without the intervention of trauma.

I have never seen an instance in which there has been satisfactory

evidence of a single mechanical trauma converting a benign tumor into a malignant one. Many times, however, evidence appears sound that benign pigmented nevi may be converted into malignant melanomas by single or repeated injuries of various sorts. To prove aggravation of a preexisting

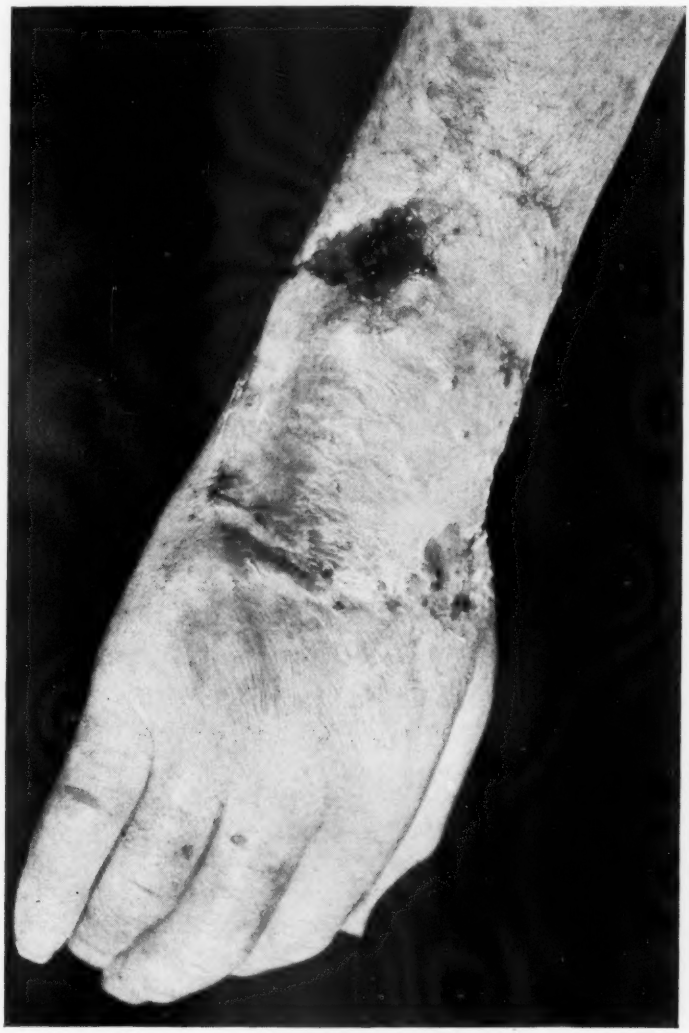

FIG. 2.—Multiple keratoses and epidermoid cancer developing in a coal-tar worker following some 30 years' exposure.

tumor by injury, it is, of course, necessary to establish that the tumor existed prior to the injury, that perceptible damage was done to it by the injury, and that the tumor's subsequent course was accelerated over that which would be ordinarily expected.[11] These factors can be evaluated only by detailed clinical and pathologic study of each case. Some evidence exists that mechanical trauma to a malignant tumor will facilitate its in-

vasion of adjacent structures and the escape of cells to establish metastases elsewhere.

To summarize, a single trauma rarely, if ever, causes a cancer, in spite of many case histories purporting to establish that fact. Repeated trauma may, rarely, produce a cancer, as may trauma to a scar. Trauma may aggravate a preexisting tumor.

SECTION II—OCCUPATIONAL TUMORS

Occupational tumors are those neoplasms that arise as a result of contact with some exogenous agent, physical or chemical, brought about by some phase of the regular work of the individual concerned, that leads to an independent proliferation of cells.

The soundest criterion for the occupational character of a neoplasm is the proof of the occurrence of a tumor of a particular type, and in a particular portion of the body, among the workers of a given industry significantly more frequently than in the general population of comparable age and sex.

As secondary proof of the occupational character of a tumor, the production of tumors in experimental animals by the suspected agent, is of great value, but it is not a prime requisite. It must be remembered that the susceptibility to various carcinogenic agents of different animal species, and even of individuals within the same species, is not at all uniform, and it is quite possible that a concentration of a given substance which is carcinogenic for man may be incapable of producing a tumor in the experimental animal. However, at times the statistical evidence that can be obtained relative to the incidence of a certain type of tumor in a certain occupation may be inconclusive and here the experimental reproduction of the tumor is of great value. In addition, of course, successful production of the tumors in animals greatly facilitates the development of preventive measures.

So far, much of the information as to the incidence of cancer in the general population is derived from mortality statistics which not only leave something to be desired from the standpoint of accuracy of diagnosis on the one hand, but give rather indefinite information as to the morbidity of the disease on the other hand. From the occupational standpoint, one is concerned primarily with cancer morbidity rather than with cancer mortality. It is to be hoped that with the passage of time an adequate mass of data as to the number of cancer cases in the general population may become available.

Sometimes it is not enough to know that an individual works in a certain general field. Thus, an oil worker may come in contact with Scotch shale oil, which has a marked tendency to produce occupational skin cancer;[1] with paraffin base petroleum oils, which are rarely associated with cutaneous malignancy;[2, 3, 4] or with asphaltic American crude oils, which have almost never produced a skin cancer. It is, therefore, important to know the exact nature of the occupational exposure.

Occupational tumors must be sharply distinguished from tumors which

arise spontaneously in employed individuals and have no relationship to their work. At times, the decision of the court as to whether a tumor is related to occupation is determined by the vigor and keenness of presentation of the picture in favor of plaintiff or defendant, rather than on a scientific analysis of the actual facts at issue. Unfortunately, at the present time the compensation laws in different states and also in different countries, relating to occupational tumors, vary a good deal, and, hence, are inequitable and often inadequate.

One factor which renders it particularly difficult to determine whether or not a given tumor is related to occupation is that often a long time interval may elapse between the onset of symptoms of the tumor and the exposure that occurs. This latent period may be only a matter of a few months or it may be many years. The existence of this latent period has not been adequately recognized in the framing of many of the industrial compensation laws, some of which contain time clauses, often of one or two years only. Legislation of this type is obviously unfair, as individuals being exposed to radioactive materials or betanaphthylamine at the present time may not show evidence of significant change for several years.

Another complicating aspect of the problem is that during the latent period the worker may change his occupation, and the individual who develops leukemia following exposure to benzol, or osteogenic sarcoma following the inhalation of radioactive material, may have changed occupation before the symptoms of the disease have become apparent, and may be employed in other occupations, such as building or office work, when the signs and symptoms of the disease become apparent.

Still another problem in some of the compensation laws is that specific chemicals are mentioned by name as recognized sources of occupational tumors, whereas closely allied chemical substances which may also give rise to these tumors are not named. It is wise to avoid overly specific cataloging of possible carcinogenic chemicals when framing or revising compensation laws.

The tumors formed as a result of agents encountered in the course of occupation affect a wide variety of tissues and, unfortunately, the majority of these tumors are malignant. A useful classification of occupational tumors is that given by Hueper,[5] a modification of which is presented herewith:

1. Direct Contact Tumors:
   a. Cutaneous neoplasms caused by direct local action of mineral oil, crude paraffin, creosote, anthracene, solar rays, ultraviolet rays, roentgen rays, rays from radioactive substances upon the cellular components of the skin.
   b. Pulmonary tumors resulting from the inhalation of radioactive material, chromates, asbestos, nickel carbonyl, tarry substances.
   c. Tumors of the nasal passages and sinuses following upon the exposure to radioactive substances, chromates, nickel carbonyl.

d. Neoplasms resulting from impingement of roentgen or other radiation on the deeper tissues.

2. Excretory Contact Tumors:
   a. Tumors of the urinary tract resulting from an exposure to certain aromatic amines excreted in the urine.
3. Depository Contact Tumors:
   a. Cancers of the skin associated with the deposition of arsenicals in the cells of this organ.
   b. Sarcomas of bone, leukemia, and leukemoid reactions following the storage of radioactive material in the bones.
4. Tumors in Tissues as a Result of Parasitic Infestation:
   a. Cancer of the bladder following bilharziasis.

The first clearly established occupational tumor is the chimney sweeps' cancer that was reported by Percival Pott,[6] in 1775. Here we have an example of a skin cancer developing in an unusual site (the scrotum) with surprising frequency in men following a particular occupation in a particular region (the British Isles).[7] It is interesting in this connection that chimney sweeps on the continent of Europe have not shown a high incidence of scrotal cancer similar to the English workers. This difference is due to the presence of a specific inciting factor in the type of coal that was burned in England as distinguished from that burned in Belgium or Germany, for example.

From these observations of Pott, interest in coal-tar as a possible inciting agent of tumor continued for many years. This reached its culmination in the isolation from coal-tar of a specific group of chemical products known as the carcinogenic hydrocarbons. While, as yet, their carcinogenic activity for man has not been proved, they are highly carcinogenic in certain of the animals.

The first experimental cancer produced by chemical means was the skin cancer of the ear of the rabbit produced by Yamagiwa and Itchikawa,[8] in 1915. Thus, the experimental proof lagged more than one hundred years behind the statistical proof of the occupational character of chimney sweeps' cancer. In many other fields, however, chiefly because of the greater interest in the experimental method and the facilities for experimental work available during recent years, the time between statistical establishment of the occupational character of the disease and experimental evidence of its relation to any specific substance has been considerably lessened. Thus, Martland[13] established, within a short time, the occupational character of the osteogenic sarcoma developing in radium dial painters.

The skin is by far the most frequent site of occupational neoplasms. This is to be expected in view of the very wide range of agents to which the skin is exposed, as contrasted with other of the tissues of the body. An important factor in the development of certain skin cancers is the amount of pigmentation that the skin contains. In general, the most heavily pigmented

skins are those which are least susceptible to damage from sunlight or other forms of radiant energy.

The mule spinner's cancer also deserves special mention. This is, again, one of the long recognized occupational tumors, and develops as a result of long exposure to oil, usually that derived from shale. Henry[7] gives the longest period of exposure prior to onset of this skin cancer as 63 years, and the shortest 16 years.

In practically all of the occupational cancers of the skin there is a precancerous phase during which the surface epithelium is heavily keratinized and has a rather marked tendency toward the development of warts or papillomata. Frequently the openings of the hair follicles and sebaceous glands may become obstructed and active proliferation may occur in the deeper epithelial cells. There is frequently a lymphocytic infiltration of the underlying corium and the line of demarcation between the corium and the overlying epithelium becomes indistinct. Sometimes the cancers arise from foci of small, atypical epithelial cells within an otherwise not remarkable skin.

Several other types of occupational cancer deserve special mention. The cancers of the bladder developing in those working with aniline and certain of its derivatives became established as an occupational disease,[9] in 1883. These cancers of the bladder develop as a result of the excretion of the inspired material through the kidneys and the prolonged persistence of the injurious agents in the urine held in contact with the bladder wall. Tumors of the bladder (both benign papillomas and cancers) are not only much more frequent in workers exposed to this group of compounds but develop in them at a younger age than in the general population. Thus Hueper[5] states that over half of the number of cases of aniline tumors of the bladder develop before the age of 50, much earlier than in those not exposed to aniline and its products. The bladder tumors may develop after a relatively short exposure to the injurious agents.[5] Thus, out of 79 malignant tumors of the bladder that developed in a group of workers, eight appeared during the first five years, and 21 during the first ten years of exposure.

Careful clinical observation and experimental work have established that many of the aniline compounds are harmless, whereas a few, as betanaphthylamine, have marked potency in producing bladder cancer.

One of the best publicized occupational tumors is the radiation cancer of the skin, from which so many of the pioneer workers with radium and roentgen ray died.[10, 11] Fortunately, we now know enough about the character of the disease to afford protection. Repeated minor exposures to the short wave radiant energy gradually bring about pathologic changes in the skin which lead to ulcerations and keratoses; later these nonmalignant lesions take on definitely malignant characteristics. The occupational character of radiation cancer of the skin was recognized early, the first occupational cancer due to radiation being reported within seven years after the first clinical use of the roentgen ray.[12]

Certain tumors may arise in greater frequency among certain occupational groups, but are not properly considered occupational because not peculiar to that particular group of workers. Thus, the farmers in the South apparently have a higher incidence rate of skin cancer than is to be expected for a corresponding age and sex group of the entire population. However, since exposure to sunshine and weather is a factor common to all, varying only in degree, it is hardly proper to consider these skin cancers as truly of occupational origin.

Another disease, perhaps not properly occupational, is bilharziasis of the human bladder. It has been known for thousands of years that the schistosomes are peculiarly apt to involve agricultural workers when exposed to infected mud and water, in those regions where the parasitic infestation is prevalent. Conditions of the Nile delta are apparently ideal for the development of this infestation, and it may properly be considered as an occupational disease of farm workers in such localities. Larvae penetrate the skin from infected mud or water or penetrate the mucous membrane of the gastro-intestinal tract after the drinking of infected water. The larval worms reach the liver and come to adult development there. They then migrate to the venous plexuses of the pelvis, especially those of the bladder wall. Some of the eggs penetrate the mucous membrane and lie within the bladder or the rectum. Their presence leads to a chronic inflammatory reaction in the bladder wall, not infrequently with patches of leukoplakia. From this injured epithelium papillomas and cancers may arise.

In general, the mesenchymal tissues are relatively well protected against the access of the various chemical factors. As a result, none of the numerous chemical substances which may induce epithelial tumors in human beings produce either sarcomas or benign tumors of mesenchymal origin. However, it must be remembered that this apparent immunity probably rests not so much on the characteristics of the connective tissue cells themselves as on their protection from prolonged exposure.

Since Diesel engines have come into more common use, the forcing of oil deep into connective tissue and muscles by the high pressure spray is not unusual, and it will be intensely interesting to see whether, with the passing of years, some of these lesions develop neoplastic tendencies.

Undoubtedly, if we can judge fairly from our past experience, there are at present in use in industry compounds which will prove in later years to have been carcinogenic. It is of the utmost importance that every precaution be taken to protect workers from appreciable contact with substances other than those proved to be innocuous. Those workers in particularly hazardous fields, such as radium dial painters,[13] should be carefully followed by all means of protection possible, and, in addition, frequent checks should be made of the actual conditions existing in the plant, both as to the hazards which exist under normal circumstances and those which may exist through some fortuitous chain of events.

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MINIMAL CRITERIA REQUIRED TO PROVE PRIMA FACIE CASE OF TRAUMATIC ABORTION OR MISCARRIAGE*

AN ANALYSIS OF 1000 SPONTANEOUS ABORTIONS

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FOR YEARS the problem of traumatic abortion and miscarriage has interested obstetricians, baffled lawyers and vexed insurance companies. These three groups are brought together in the courtroom to see that justice is served in those cases involving trauma, either physical or psychic, which is alleged to have caused the premature expulsion of a nonviable fetus. It is the consensus of opinion, of at least the average conscientious expert medical witness involved, that, all too often, justice is not served and that the plaintiff is awarded damages for an abortion or miscarriage in which the trauma was, at most, only coincidentally concerned. It is the purpose of the present communication to examine and put forth the factual relationship between trauma and abortion or miscarriage.

Medically speaking, abortion is defined as the premature expulsion of a nonviable fetus. Broadly speaking, however, the definition should substitute the word "ovum" for "fetus," since, as will be shown later, almost one-half of the abortuses in the author's series contained no fetus at all; the products of conception consisting either of an empty ovisac or one occasionally containing an unformed embryonic rudiment.

To the average layman, an abortion means the termination of a pregnancy by artificial means, either criminally or self-induced. A miscarriage or "miss" on the other hand, connotes a spontaneous termination of a previable pregnancy. To complicate the terminology still further, the average medical person regards an abortion as covering the period up to the sixteenth week of gestation and a miscarriage as covering the period from the sixteenth to the twenty-eighth week of gestation, the average period at which the fetus becomes, theoretically, viable. It is clear, therefore, that the term abortion, when used in the broadest medical sense, includes all previably delivered ova although in a more restricted sense refers to only the first 15 weeks of gestation. These factors of terminologic confusion must be kept in mind by all three of our interested groups when conferring, either with one another or with the lay public. Hereinafter the term abortion will be used in the broadest medical sense.

That there is a real relationship between trauma and abortion is ad-

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mitted by all qualified medical experts in the field (Taussig[1]). However, the number of *bona fide,* traumatically caused abortions is much rarer than is generally supposed by the average lay, legal and medical public. It is proposed to cite the evidence for this relationship by analyzing a series of abortuses examined in the Pathological Laboratory of the Boston Lying-in Hospital, either by or under the immediate supervision of the author.

MATERIAL AND METHODS

The period encompassed in the study is from February, 1936, to December, 1941. During this six-year period a total of 1416 consecutive abortuses were carefully examined from an embryologic as well as a pathologic point of view. Many of these 1416 cases have been submitted by physicians in Boston, although moderate numbers of specimens have been received from hospitals and physicians in other communities. Since a good many of the physicians on the staff of the Boston Lying-in Hospital submit every spontaneously aborted ovum from their private practices, it is felt that this material, with the specific exceptions of hydatidiform moles and criminal abortions, is representative of the variety of pathologic conditions associated with spontaneous abortion in general.

Of these 1416 abortuses, 1000 were selected as the basis of the present report. The criteria of selection are based merely on the completeness of both the clinical history and the pathologic material submitted. Obviously, an incomplete abortion, containing only a tiny bit of curetted decidua and a few chorionic villi is not sufficient material upon which to base valid conclusions for a study of this sort. Furthermore, a series of 63 hydatidiform moles examined in the laboratory during this period are excluded from the present study since they would seriously invalidate the statistical value of these data. Such moles are rare (approximately 1:2000 cases of pregnancy) but are prominent in the total series because the author is especially interested in that subject and has collected them from all over the country. The 1000 selected cases, therefore, represent a consecutive series of abortuses which are complete enough, both as to history and material, so that valid conclusions may be drawn from their study. The round number chosen also facilitates figuring percentage values. The general technic used in the examination of this material is detailed in Mall and Meyer's[2] classic work reporting the pathologic ova in the first 1000 accessions to the world-famous embryologic collection of the Carnegie Institution of Washington.* In 1940, the author[3] published certain data from the first 1027 abortuses examined at the Boston Lying-in Hospital pertaining to the genesis of hydatidiform mole. Detailed notes on the technic of examining these abortuses are given in that paper.

* It was the privilege of the author to spend a year in the Department of Embryology of that Institution under the guidance of Dr. Franklin P. Mall's successor, Dr. George L. Streeter, to whom the author is greatly indebted for instruction in the methods of studying normal and abnormal human ova. For the past nine years, since the initial year of study there, the author has collaborated in the work of that laboratory, now under the direction of Dr. George W. Corner.

Since it is the consensus of medical opinion that at least ten per cent of all pregnancies terminate in spontaneous abortion, and that only rarely are such abortions caused by trauma, the main emphasis in this paper will be to point out the apparent primary etiology in 1000 abortions and to discuss in detail the relationships of trauma to abortion in the 13 cases of this series in which trauma of any sort antedated the abortion. It should be noted here that in only one of these 13 cases was external trauma, an automobile accident, responsible for the abortion. In view of the frequency of spontaneous abortion and the rarity of true traumatic abortions, it becomes of the greatest importance to evaluate mere coincidental trauma in spontaneous abortion. The thesis of this report, therefore, may be summed up by stating that, in the opinion of the author, the plaintiff in a case of alleged traumatic abortion must present proof of the presence of a normal pregnancy at the time of the trauma and that the abortus shows objective clinical, embryologic and pathologic evidence of the relationship to the trauma.

MAIN CLASSIFICATION OF MATERIAL

The classification followed in studying these abortuses is essentially the same as that devised by Mall (Mall and Meyer[2]). Pathologic ova in Mall's original classification are those in which the embryos are absent, very defective or macerated. The author has modified this classification by excluding the macerated embryos from the pathologic group since, broadly speaking, these embryos are anatomically normal but have died in utero for one reason or another. Hence, in this series many so-called normal ova contain macerated embryos although the ovisacs show various pathologic changes which account for the death of the embryo and subsequent abortion of the ovum.

The following figures are a crude breakdown of the 1000 cases with respect to the main embryologic and pathologic features shown in each specimen, although each subgroup will be subsequently discussed more in detail:

I, OVULAR FACTORS:		
1. Pathologic ova, with absent or defective embryos	489	
2. Embryos with localized anomalies	32	
3. Placental abnormalities	96	
		617
II. MATERNAL FACTORS:		
1. Criminal abortions	21	
2. Uterine abnormalities	64	
3. Febrile and inflammatory diseases	20	
4. Miscellaneous	12	
5. Anatomically normal ova (classified)	265	
6. Trauma (automobile accident)	1	383
		1000

I. OVULAR FACTORS

1. *Pathologic Ova*

These specimens constitute the largest single group in the series (48.9 per cent) and are the typical "blighted ovum" of the clinician. They are classified in more detail as follows:

Group I.—*Villi Only:* This material contains only chorionic villi, whether normal or abnormal. Obviously, this group is one of convenience only, since it merely classifies the material submitted, which may or may not adequately represent the relationship between maternal and ovular tissues. This group, therefore, includes curettings from cases of incomplete abortions. Actually very few of the large number of such cases in the total series of 1416 abortions are included in this study because of the obvious incomplete nature of the material.

Group II.—*Empty Chorionic Vesicle:* This type of specimen when intact (as it often is) represents the most pathologic type of ovum with which the pathologist has to deal. There is no derivative of the inner cell mass; that is, of the portion of the fertilized ovum destined to form the embryo. If the chorion is ruptured, one might have reasonable doubt about the essential pathologic nature of the ovum; that is, the normal embryo with its surrounding amnion may have been lost during the abortion. However, if trauma has produced such an artefact in an otherwise normal ovum, one can usually see evidence of the torn stump of a normal umbilical cord with its radiating vessels.

Group III.—*Chorion Containing Empty Amnion:* This type of ovum is only slightly less pathologic than the previous one, there being no evidence of an embryo, although the amnion is present. Members of this group are, likewise, valid if intact—as they often are—although if ruptured, erstwhile normal ova with the embryos missing can usually be detected and differentiated from true Group III specimens.

Group IV.—*Chorion and Amnion Containing Nodular Embryo:* This type is truly pathologic, as the embryonic mass consists merely of a disorganized group of embryonic cells. Artefacts in this group would consist of the macerated remains of an otherwise normal umbilical cord within either a ruptured or intact amnion.

Group V.—*Chorion and Amnion Containing Cylindric Embryo:* If the head end of the embryo can be recognized, even though it does not possess any other features of an embryo, such a specimen is valid for this group. (It is quite rare in the author's experience.)

Group VI.—*Chorion and Amnion Containing Stunted Embryo:* It is possible to recognize the embryonic form, although it is much smaller than it should be for the menstrual age of the specimen. In addition, one or more portions of the embryo are atrophic, deformed or degenerated. These embryos are usually not macerated. This is a valid group whether the chorion and amnion are ruptured or not, since the embryo has to be recognized before the specimen may be placed in this category.

The vast majority of this large and important group, destined for abortion, whether there is associated external trauma or not, belongs in Groups II, III and IV. They tend to abort during the tenth week of gestation. Evidence recently accumulated by the author,[4] in collaboration with Dr. John Rock, show that these pathologic ova are deficient from the early stages of development before the patient has any knowledge that she is pregnant.

2. *Embryos with Localized Anomalies*

These specimens constituted 3.2 per cent of the total series. The congenital anomalies include many of the common deformities of the nervous system such as spina bifida, meningo-encephalocele, anencephaly, *etc.*, which may go to term but are incompatible with continued extra-uterine existence. Other anomalies are also found in the group such as deformed extremities. That these particular anomalies are responsible for the death of the embryo and subsequent abortion of the ovum is not rigidly maintained. It is significant, however, that the incidence of congenital anomalies is 4.7 times greater in this series than in Murphy's[5] series of stillborn and living fetuses which went to term. Hence, the presence of a congenital anomaly in an aborted fetus must be *prima facie* evidence of some general ovular abnormality which is expressed by the presence of the localized anomaly seen in the specimen.

3. *Placental Abnormalities*

This relatively large group constitutes 9.6 per cent of the total. Its inclusion in the main group of abortions due to ovular factors may be questioned since the etiology of the single largest group, the circumvallate placenta, is not entirely clear. However, since the circumvallate placenta, which tends to separate prematurely and, hence, causes abortion and premature labor, is an abnormal placenta, whatever its cause, it is included here under the general heading of abortions due to ovular factors.

The general group of placental abnormalities, together with the number of cases of each variety are listed as follows:

a. Circumvallate placenta	45
b. Hypoplasia of the placenta	20
c. Placenta membranacea, partial	2
d. Velamentous insertion of umbilical cord	1
e. Hypoplasia of amnion	1
f. Rupture of marginal sinus	3
g. Premature senility of placenta	4
h. Berus' mole (intraplacental hematomata)	19
i. Succenturiate lobe with total infarction	1
Total	96

The common denominator with respect to the cause of the abortion in most of this group is death of the fetus and subsequent expulsion of the ovum. Some of the circumvallate placentae, when they separate prematurely at their margin, are associated with premature labor, in which case the embryo is still living when delivered.

II. MATERNAL FACTORS

1. *Criminal Abortions*

This group is necessarily small and constitutes 2.1 per cent of the total. It is expected that criminally induced abortions would not be numerous in a group of spontaneous abortions submitted to the pathologist in an attempt to discover the cause of the abortion. However, the author has been interested in the pathologic sequence of events in criminally induced abortions and has attempted to acquire material, thus accounting for the few cases of this type among the group of otherwise spontaneous abortions. The figure of incidence, therefore, is entirely erroneous and much lower than actually exists throughout the country. Suffice it to say that the pathologic picture is often one of artificial premature rupture of the membranes followed by acute bacterial inflammation of the chorion, amnion, placental villi and decidua.

2. *Uterine Abnormalities*

This is a distinct group constituting 6.4 per cent of the total. The various subdivisions of this group, together with the number of cases of each are as follows:

a. Low implantation of placenta	56
b. Placenta accreta	2
c. Bicornuate uterus	2
d. Multiple leiomyomata of uterus	1
e. Retroversion of uterus, fixed	3
Total	64

It may be questioned as to why the low-implanted placentae (including five definite cases of placenta previa) should be considered as due to any maternal factor. The reason for assuming this, as yet unproved, relationship is because of the high incidence of placenta previa in multiparae. It seems unlikely that the fertilized ovum of a multigravida is any different from that of a primigravida although the postpartum uterus of the former never quite involutes back to its nulliparous state.

3. *Febrile and Inflammatory Diseases*

This small but important group constitutes 2 per cent of the total series and includes local bacterial inflammation of the endometrium, both acute and chronic as well as febrile states from various causes. The cases are listed as follows:

a. Bacterial inflammation of decidua, acute	12
b. Small pox	1
c. Pyelitis with horseshoe kidney	1
d. Fever of unknown etiology	5
e. Chronic endometritis	1
Total	20

It is worthy of comment that local inflammation plays a relatively small part in the etiology of spontaneous abortion. This is in contrast to the views advanced by Mall and Meyer,[1] who ascribed to inflammation a prominent rôle in the causation of abortion. It would appear that they misinterpreted the leukocytic response to sterile decidual necrosis following thrombosis of sinusoids—a universal finding in all spontaneous abortions of whatever etiology.

4. *Miscellaneous*

This small and heterogeneous group (1.2 per cent, is formed as a matter of convenience only. There is no relationship between any of the following group of cases:

a. Radiation effect on ovaries	2
b. Erythroblastosis fetalis	3
c. Surgical removal of corpus luteum	4
d. Blood dyscrasia	1
e. Interference with circulation of cord	2
Total	12

5. *Anatomically Normal Ova (classified)*

This is the next largest group in the series and constitutes 26.5 per cent of the total. These abortuses all have one factor in common; namely, an anatomically normal ovum to which various things have happened *in utero*. The following tabulation gives the main subdivisions of this large and, in general, unsatisfactory group:

a. Anatomically normal ova without disease		227
Fetus, macerated	146	
Fetus, nonmacerated	74	
Fetus, by history only	7	
	227	
b. Acute chorionitis, consistent with spontaneous premature rupture of membranes (fetus macerated in 9 and normal in 5)		14
c. Positive Hinton and Wassermann tests		3
(The syphilis was probably not responsible for the abortion)		
d. Infarction of placenta, extensive		13
(All fetuses were macerated)		
e. Toxemia of pregnancy		5
(Fetus macerated in 3 and normal in 2 cases)		
f. Trauma (internal)		1
(Two successive biopsies on sterility patient not known to be pregnant)		
Exploratory celiotomy		1
Intrauterine lipoidal injection, 7 weeks prior to last menstrual period		1
Total		265

It is obvious that the large number of cases (227) in group "a" show no satisfactory

cause for their abortion. That the placenta prematurely separated in some of the cases (27) and that the patient then went into premature labor is only begging the issue. Of course, nearly two-thirds of this group showed macerated fetuses, a perfectly adequate cause for the abortion but the cause of the intra-uterine fetal death is still obscure.

Those cases in group "b" who prematurely rupture their membranes with subsequent infection of the ovisac and expulsion of either a dead or living fetus are, in the last analysis, unexplained. What causes the membranes to rupture prematurely is unknown, although it is the impression of the author that the ovisac is too small in many cases as compared to the size of the growing fetus.

It is interesting to note the relative paucity of cases complicated by syphilis. In older dissertations on abortion, syphilis was prominently mentioned as a cause but at the present time it would seem to play no etiologic rôle in spontaneous abortion.

Toxemia of pregnancy is uncommon during the period of gestation concerned in the cases in this study. It may kill the fetus directly, by means as yet unknown, or it may cause fetal death by toxic premature separation of the placenta.

The one case of internal uterine trauma, aside from those in the criminally induced group, is of interest from a medicolegal point of view. It illustrates that extreme care must be exercised in doing endometrial biopsies on sterility patients, especially after the period of implantation of the ovum. Since most, if not all sterility patients may theoretically be pregnant during the last half of the menstrual cycle, it is possible that a biopsy performed after the 19th to the 22nd day may mechanically interrupt a pregnancy which, as yet, necessarily, gives no evidence of its presence.

The second case in group "f," the abortion following an exploratory celiotomy, is of interest. The patient had been widowed for three years (a factor of probable importance in this case). Since the last menstrual period was not known, or admitted by the patient, and because the periods were alleged to have been regular but scanty, the enlarged uterus was thought, clinically, to have been a fibroid uterus. The patient was flowing at the time of the exploratory celiotomy, which revealed a normal pregnant uterus of approximately 3½ to 4 months gestational age. A week following the operation the patient miscarried a normal but macerated fetus of 16 weeks gestational age. The cord was about the neck twice, although this was probably not of etiological significance in the death of the fetus since a dead fetus may move about passively in the uterus due to uterine contractions and general activity on the part of the patient. Whether the operation caused the abortion cannot be proven. There are no data by which this factor can be judged since the menstrual age of the pregnancy could not be accurately ascertained. In view of an atypical story by a widow of three years standing, who was bleeding at the time of operation, it is justifiable to wonder, at least, if this is not a traumatic abortion of the self-induced or criminal type rather than due to the operation.

The final case in group "f" is also of medicolegal significance in that the sterility patient had a lipiodol injection into the uterus and tubes seven weeks prior to the last menstrual period before she became pregnant. She aborted a normal but macerated eight weeks fetus and chorion at ten weeks and five days after the last menstrual period. It is difficult to see what relationship, if any, the lipiodol uterotubogram had on the subsequent abortion unless one postulates that the oily substance, which is known to persist for some time in the tubes, affected the fertilized ovum on its passage through the latter. However, the fertilized ovum was of such vitality that it developed to the stage of a normal eight weeks pregnancy at which time the embryo died. It seems unreasonable to assume that the theoretically unfavorable environment created by the lipiodol persisting for seven weeks in the tubes, could have had a delayed effect on the ovum. It is well recognized among teratologists that the developing ovum is affected relatively soon after its exposure to unfavorable environments. However, one cannot absolutely rule out the delayed effect of lipiodol and so the case is included in this general group.

6. Trauma (automobile accident)

This single case of a normal twin pregnancy which aborted following, and because of the patient's involvement in, an automobile accident is a classic example of a *bona fide* traumatic abortion. It satisfies the criteria reserved for such cases by the conscientious medical expert and is, therefore, deserving of a detailed history.

This patient was a 22-year-old gravida I, para O, whose last menstrual period occurred on November 24, 1937, 12 weeks and two days prior to her involvement in an automobile accident. This occurred while sitting in a stationary automobile which was struck from the rear by another car. The patient was excited and emotionally upset, although there was no mention in the clinical history of definite bodily injury. Seven hours after the accident the patient began to have painful uterine cramps followed in five hours by slight vaginal bleeding. Morphine, grains ½, given in two divided doses one hour apart, failed to alleviate the uterine pain which, after six hours, became steady and was localized in the right lower quadrant. The patient experienced a chill at this time but had no elevation of temperature. She vomited once. The pulse was 82 and the blood pressure 120/60. Physical examination, performed by an obstetrical consultant, showed a nontender pregnant uterus of approximately four months gestational age. The uterus was, therefore, somewhat larger than the menstrual history would indicate. (This was later shown to be due to an excess amount of amniotic fluid and normal twin fetuses, each of which was normal in size and development for the stated duration of pregnancy)

The patient was admitted to the hospital 11 hours after the onset of uterine pains (labor). She soon ruptured her membranes spontaneously and delivered normal twins an hour later; 19 hours after the accident and 12 hours after the onset of labor.

Pathologic examination revealed an anatomically normal twin pregnancy whose fetuses each showed the expected degree of development for their menstrual age (12 weeks and three days). The menstrual age of aborted embryos is determined by reference to tables of embryonic development compiled by Dr. George L. Streeter,[6] recently retired director of the Carnegie Institution of Washington's Department of Embryology.

There is no reasonable doubt but what the accident initiated labor. Whether it did so by means of direct trauma or the psychic shock incident thereunto is impossible to say. It is quite possible that the increased distention of the uterus due to the twin pregnancy, made the organ more irritable and hence more susceptible to the factor or factors which initiate labor. Be that as it may, it would appear that this is a case of *bona fide* traumatic abortion for which the patient should, with justice, be compensated. It is of interest that as the result of this accident the patient received $700 to $800 in addition to having her hospital bill paid.

ABORTUSES WITH A HISTORY OF ANTECEDENT TRAUMA

In the entire series of 1000 abortions concerned in this study, there are nine cases in which external trauma of some variety was recorded prior to the expulsion of the ovum. Five cases proved, on pathologic and embryologic examination, to have passed pathologic or defective ova which would undoubtedly have aborted irrespective of any trauma, while four cases showed placental defects which caused the abortion.

Four of the patients who passed pathologic ova had taken long automobile rides a week or ten days prior to the abortion. Inasmuch as patients are warned against this practice, it is of some interest to note that, at least in these four cases, the automobile ride bore no causal relationship to the

abortion; none of the ova contained any embryo (except a nodular one in one case) and, hence, all were destined to abort.

The fifth case, involving a pathologic ovum, is deserving of special comment because the patient experienced a back strain, a fall and a death within the family prior to her passage of a typical blighted or pathologic ovum. The latter consisted of the most pathologic variety encountered—the empty chorionic shell without vestige of amnion or embryo. Embryologically, this ovum had been abnormal prior to the ninth or tenth day of its development, at which time the amnion begins to form. It is obvious that none of these various forms of trauma, including the psychic shock of the death in the patient's family, had anything to do with the subsequent abortion.

The four remaining cases in which the abortuses showed placental defects can be discussed as a group. The traumata associated with these cases are as follows: a long automobile ride just before abortion, psychic shock or trauma suffered as the result of being near an automobile accident two days prior to abortion, involvement in an automobile accident six weeks prior to abortion and a severe fall at home two days prior to abortion. All pregnancies progressed normally until the 19th to 20th weeks. All patients passed normal fetuses, two of which were macerated, one was normal and one was not submitted although it was said to be normal. The placentae all showed varying degrees of circumvallate formation; a developmental defect of the placenta, probably associated with too shallow implantation of the ovum in the endometrium. This defect of implantation, with its resulting poor maternal blood supply, allows only a relatively small area of the chorion, or external shell of the ovum to form a placenta. In order for this small patch of placental tissue thus formed to supply the fetus with sufficient food and oxygen, the lateral margin of the placenta grows radially and in so doing causes premature separation of the placenta itself.

Inasmuch as implantation of the fertilized ovum occurs on the nineteenth to the twenty-second day of the menstrual cycle,[4, 7] or about a week prior to the patient's knowledge of her pregnancy, it is obvious that trauma occurring after pregnancy has been established cannot be responsible for abnormalities of the placenta due to faulty implantation of the ovum.

DISCUSSION

From the foregoing data it is clear that antecedent external trauma or psychic shock may appear in the history of a case of abortion. That trauma is only rarely etiologically related to the abortion is also evident from an analysis of the 13 cases in which antecedent trauma is recorded; only one of these abortions was caused by external trauma. When trauma is an etiologic factor in an abortion it must immediately precede, by a matter of hours, the onset of the sequence of events that results in an expulsion of a normal ovum.

It is obvious, furthermore, that in the case of a *bona fide* traumatic abortion, the ovum must be shown to be developing normally up to the time at which

the trauma occurred, since there are many other factors independent of trauma which can and do cause abortion. Since true traumatic abortion is such a rarity and spontaneous abortion such a common occurrence, even though antecedent trauma may be recorded in proven spontaneous abortions, it is mandatory that the normality of the pregnancy be proven in cases of alleged traumatic abortion. In the light of the findings in this series of abortions, it becomes obvious that the normality of a given pregnancy cannot be determined by clinical examination alone. The normality of the ovum can be proven only by embryologic and pathologic examination of the abortus by someone expert in this special field of pathology. Finally, it would seem to the author that the burden of proof of the relationship between any given trauma and the subsequent abortion be upon the person who alleges that the particular trauma caused the resulting abortion.

To verify the relationship of trauma to abortion the author consulted seven obstetrical specialists in Boston, all of whom are members or emeritus members of the Obstetrical Society of Boston. They were questioned as to the number of *bona fide* cases of traumatic abortion encountered in their obstetrical experience. The number of cases and their years in practice are as follows:

1 case in 38 years (the case reported here)
3 cases in 28 years
2 cases in 27 years
0 cases in 25 years
0 cases in 22 years
0 cases in 10 years
0 cases in 7 years

The specialists who had taken care of such traumatic abortion cases were in unanimous agreement that the onset of signs or symptoms leading to abortion followed the causative trauma within minutes to hours. The initial sign may have been either rupture of the membranes or vaginal bleeding followed by uterine cramps and expulsion of the normal ovum. The etiologic trauma took such diverse forms as extreme exertion during a severe thunder storm (1 case), automobile accidents (3 cases), climbing the mast of a sailboat during a race in order to fix a broken halyard (1 case), and a severe paroxysm of coughing due to whooping cough (1 case).

The history of one additional case has come to the author's attention recently. The patient was thrown clear of an automobile, sustaining lacerations of the right thigh and vulva together with a fractured skull. When picked up soon after the accident she was bleeding profusely from the vagina due to an inevitable abortion which was completed in the hospital. The ovum was normal.

SUMMARY

The analysis of 1000 cases of abortion, selected as to the completeness of their clinical history and pathologic material, shows that external trauma and/or the psychic shock associated with it did, in one case, initiate the

sequence of events resulting in the abortion of a normal twin ovum. Careful pathologic and embryologic examination of 11 abortuses from patients who gave a history of antecedent trauma showed adequate natural causes for the abortions—the trauma in such cases being purely coincidental. The remaining 988 abortions showed no etiologic relationship to external trauma although 21 of them were criminally induced, and one normal ovum aborted as the result of two endometrial biopsies done on a sterility patient prior to her first missed period.

CONCLUSIONS

1. A series of 1000 abortions are reported and their etiology determined by clinical history, embryologic and pathologic examination.

2. One case of a normal twin pregnancy aborted at 12 weeks and three days as the result of external trauma and/or the resulting psychic shock resulting from an automobile accident.

3. Eleven cases of abortion preceded by various external traumata are analyzed and shown to be due to natural causes.

4. The remaining 988 abortions (including 22 induced by internal uterine trauma) were due to a variety of naturally occurring ovular and maternal factors.

5. The collective experience of a representative group of Boston obstetrical specialists is reported with respect to abortions occurring as the result of external trauma and/or psychic shock.

6. A *bona fide* traumatic abortion is one in which the abortus was anatomically normal in development up to the time at which the external trauma and/or psychic shock occurred. If this predicate cannot be proven, we must regard the evidence of traumatic causation as conjectural and speculative, and suspect that the abortion was a spontaneous one due to pathologic causes.

7. Minutes to hours is the time interval between the occurrence of the external trauma and/or psychic shock which initiates the sequence of events resulting in the expulsion of a normal ovum.

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THE RELATION OF TRAUMA TO DIABETES*

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CERTAIN FUNDAMENTAL FACTS are herewith recorded, essential for a proper understanding of diabetes, and following these are various concepts which will serve as a summary of the relation of trauma to the disease.

FUNDAMENTAL FACTS CONCERNING DIABETES

1. Diabetes is an hereditary disease, characterized by an increase of sugar in the blood and the excretion of sugar in the urine; it is dependent upon the loss or decrease of the insulin secreted by the islands of Langerhans of the pancreas and is functionally interrelated with other endocrine glands, particularly the pituitary but also the adrenal, thyroid, and the liver.

2. Proof of the diagnosis of diabetes is all important. The lack of accurate, diagnostic tests in the past and the failure to distinguish between glycosuria and the disease (diabetes) renders valueless most of the older literature.

3. Diabetes is universal. It ranks eighth as a cause of death in the United States, and approximately one individual in 165 of the total population has the disease. No age, sex, race or social status is immune. Its incidence is increasing and presumably will continue to grow until the average age at death of the population exceeds the decade 44–55 years, in which it is most apt to begin. This makes the date of onset of the disease in relation to the time of the trauma a crucial factor. This is especially true if influences favoring the development of diabetes already exist. The onset of diabetes is usually indefinite, but it may be sudden and in the span of 24 hours, and was so classified by me in 1.3 per cent of one series of 7000 of my cases.

4. Legal proceedings, based upon trauma, during the course of diabetes either should be avoided or entered upon after unusual deliberation by diabetics. A diabetic may go to court and win his suit, but this discourages employers from hiring or even keeping in their employ other diabetics and puts off the day when the diabetic can enter government service or secure

* The author, with his associates, has written a more detailed discussion of Trauma and Diabetes in the volume Trauma and Disease, Brahdy and Kahn, 2nd Ed., Lea & Febiger, Philadelphia, 1941, pp. 536–589; also in The Treatment of Diabetes Mellitus, Lea & Febiger, Philadelphia, 1940, 7th Ed., pp. 76–87. In each of the foregoing articles references to the literature and citations of cases are numerous, but in this article I have tried to approach the subject from a somewhat different and more confident point of view because of the 22,258 diabetic cases having consulted my associates and me and, also, because recent advances in diabetic knowledge have confirmed many earlier suppositions. In general, the literature cited here is in addition to the bibliography of 83 or more references in the most recent of the above publications.

insurance. There are about 800,000 diabetics now living in the United States and in a peculiar sense each one is his "brother's keeper."

CONCEPTS CONCERNING TRAUMA AND DIABETES

1. The thesis that trauma *de novo* can cause diabetes has steadily lost support with the expanding knowledge of the nature of the disease.

2. But evidence has accumulated to show that trauma indirectly can activate, or accelerate the appearance of a latent diabetes in the hereditarily predisposed, particularly if accompanied by infection, reduced muscular exercise, gain in weight or overeating.

3. Trauma in the course of diabetes has grown in importance, because the duration of the disease has trebled, thus lengthening the period of exposure. Moreover, the danger of exposure to trauma is intensified each successive year a diabetic lives, because time is provided for the disabling complications of the disease to appear and the physical infirmities of the normally aging process to advance.

The tissues of a diabetic are more vulnerable than those of a non-diabetic.

4. Trauma may make the diabetes more severe, but this effect is not necessarily permanent.

5. Emotional, nervous, so-called neurogenic diabetes, as von Noorden well said, was put "into the grave" by the Great War, and there it is likely to remain unless exhumed during the present conflict.

6. To prove that trauma is the cause of diabetes in any individual case evidence must be at hand to show (a) that the disease did not exist before the trauma; (b) that the trauma was severe, injuring the pancreas; (c) that the symptoms and signs of the disease developed within a reasonable period following the trauma, the etiologic importance of the trauma waning with the prolongation of the interval; and (d) that the symptoms and signs of diabetes were not transitory but permanent.

7. This question of trauma as the cause of diabetes should be kept absolutely distinct from the question of compensation of an individual who is found to have diabetes following an accident. Too often, especially in foreign publications (Lommel, Troëll) the two are confused, and for social and governmental insurance reasons the court sitting in judgment on a case may vote to give the insured the benefit of a doubt which has no factual basis. Many European countries are saturated with social accident insurance, and if a citizen is not actually in the employ of the government, at least he expects a liberal interpretation of social or insurance benefits.

THE DISEASE DIABETES

1. *Diagnosis.*—The diagnosis of diabetes depends upon the demonstration not only of glucose in the urine (glycosuria), but also of a per cent of glucose in the blood (glycemia) of 130 mg., or above, when the subject has been without food for five or more hours or of 170 mg. (hyperglycemia),

or more, after intake of food. Insurance companies are suspicious of 120 mg. per cent fasting, and some clinicians raise the boundary line after food to 180 mg. per cent. Fifteen (14.8) per cent of all cases consulting me during the period 1897 to 1935 for a possible diabetes proved on investigation not to be diabetic. All but 32 of these 1946 cases were traced, and the diagnosis, in the course of years, was subsequently changed to diabetes, usually mild in character, in 193, but only in approximately a third of this number was it altered if the diagnosis was originally based on glucose tolerance tests. Other sugars, levulose, lactose, and pentose are occasionally found in the urine, but they have no connection with true diabetes. Glycosuria (non-diabetic), levulosuria, lactosuria and pentosuria are harmless states. The common tests for glucose in the urine are reliable and seldom subject to error, but this does not hold true for tests of the blood sugar which are more complicated and to be diagnostic must be carried out with special precautions regarding technic and reagents. The diet and the physical status of the subject at the time of the examination are of prime importance, else the reliability of the diagnosis is open to question.

Proof of the diagnosis of diabetes is all-important. Accurately planned and well-meaning conclusions concerning trauma and diabetes in the past today fall flat because the early authors did not have the facilities to distinguish between glycosuria and diabetes. Konjetzny and Weiland's conclusions, in 1915, upon glycosuria, diabetes and fractures, although still often quoted, are invalidated by the modern studies of Timpe, supported by tests of the blood sugar.

This very month a priest, Case No. 22290, told me, in applying for a Chaplaincy, he passed the Selective Service, including an examination of the urine, but two weeks later diabetes was discovered because the test was performed soon instead of long after a meal. Case No. 2063, with diabetes of 23 years standing, onset at 14 years of age, wrote me he also passed his entire physical examination including urine test, but was finally disbarred when he revealed his diabetes. He died suddenly in December, 1942, presumably of coronary thrombosis. The insurance policy which he obtained one year before was obviously cancelled.

2. *Did Diabetes Precede the Trauma?* Whenever the question of trauma as the cause of diabetes or as an incident in the course of diabetes arises, one should establish whether diabetes existed before the accident. For this purpose a rigorous search for symptoms and signs of the disease should be made as well as for the existence of factors predisposing to its development so that the date of onset can be determined with reasonable accuracy. This is not the place to discuss the symptoms and signs of diabetes, which are to be found in text books of medicine or in the monographs of Joslin, Root, White and Marble, and of Wilder, but some of the statistical data and influences provocative of the disease deserve attention.

What are the chances of an individual in the United States already having

diabetes at the time of an accident? The National Health Survey computed the number of diabetics in this country, in 1938, as at least 660,000, but in my opinion for this year, 1943, it is nearer 800,000. Diabetes occurs at any age, but with increasing proportion as one grows older. The frequency is 1 in 2500 up to 15 years of age in either sex, and reaches 1 in 70 for males and 1 in 45 for females at 65 years and above. Among Jews from early middle life on the incidence is higher, perhaps twice as great, being highest of all among Jewish women between the ages of 55 and 64, the proportion of deaths from diabetes to total deaths among Jews in New York City, in 1933, being 11.5 per cent. The draft has stimulated the finding of new cases not alone in the young but at all ages. In 1900 diabetes was 27th as a cause of death; in 1938, it was ninth, but in 1941 it advanced to eighth place. Diabetics are living longer and longer. Tabulations made by the Statistical Department of the Metropolitan Life Isurance Company, based on my own fatal cases, show that the duration of life of the average diabetic advanced from 4.9 years between 1898–1914 to 14.3 years between 1940–1942. The expectancy of life for a diabetic, also computed for my series, is about two-thirds that of the population as a whole for comparable age-groups.

A positive heredity also increases the chances of the individual having diabetes. Pincus and White conclude from their studies that at least 25 per cent of the population carry the hereditary gene for diabetes and are, therefore, predisposed to the disease. Heredity is greatest in identical twins, reaching 70 per cent; in cases with onset in childhood and living 20 or more years it is 62 per cent, and for 1800 diabetics on my service at the George F. Baker Clinic at the New England Deaconess Hospital in 1941 was 40 per cent.

A diabetic heredity makes an individual susceptible to the disease, but of all factors predisposing to its development by far the most potent is obesity. It precedes the onset of diabetes in more than three-fourths of the cases, and from 40 years onward reaches a still higher per cent. Among 2000 of my own cases of diabetes, not one occurred who was more than 30 per cent underweight, and in Adams' series of 1000 cases at the Mayo Clinic, no patient developed the disease who was more than 20 per cent underweight.

The type of onset of diabetes is of great significance no matter whether it is indefinite in 85 per cent of the cases, or sudden, within the space of 24 hours, in a trifle over one per cent, as already mentioned. Whereas the slow, unobtrusive onset presents many a puzzling problem before an approximately correct date can be fixed, the cases of sudden onset offer the best evidence for a *post hoc propter hoc* argument. Eight thousand (one per cent of 800,000) or more cases with a sudden start of their disease are now available in the United States. What opportunities they afford for traumatic, diabetic exploitation! This special group represents the *élite* corps from which recruits for the traumatic etiology of diabetes should be most easily obtained, but that I have recognized none with a traumatic basis among the

more than 200 of this class I have personally studied, is of some import. One of the best examples in my own clientele of the sudden beginning of diabetes is the following:

Case No. 13,332, who was in perfect health, so far as he or his family knew, on December 24, 1934. That night, this 14-year-old Jewish boy, with diabetic heredity, slept without rising from bed. On Christmas day there was no especial excitement or careless eating, yet at night he rose six times, and 17 days later, when I first saw him, the urine contained eight per cent sugar. He was doing well in September, 1942, but the diabetes persists.

There was no accident here to cause diabetes. There was no heredity known at the time, although later it was learned his mother's cousin had diabetes. Suppose your automobile injured this lad while coasting on Christmas Day, or that for some reason you had severely reprimanded him and he had undergone an emotional reaction. I feel confident that abroad, if the case came into court, the acute onset of his diabetes would be put in evidence against you, and for social and insurance reasons he would have secured favorable consideration unless in a country where he would be debarred by race. In such a situation attention should be focused on the overwhelming number of similarly acute cases, which spring into being without any reason whatsoever to suspect physical or psychologic trauma.

All these considerations, therefore, show how essential it is to investigate the background of any case of trauma in which diabetes may be a factor.

TRAUMA RARELY A DIRECT CAUSE OF DIABETES

Less and less credence has been given to the direct causation of diabetes by trauma since 1889, when it was demonstrated that its etiology and pathology centered in the pancreas. Total removal of the pancreas in a dog invariably brings on the disease, but that organ has such a high factor of safety that it fails to appear if more than one-fifth of the gland remains. This anatomic fact, in itself, shows how futile the attempt must be to connect bodily trauma with diabetes. Moreover, the pancreas lies in the depths of the abdominal cavity, with the abdominal wall, peritoneum and stomach in front of it, is partly overhung by the liver and in close touch with the spleen, and posteriorly is protected by the backbone. It is hardly conceivable that four-fifths of it could be destroyed and yet allow life to go on. The discovery that insulin, the hormone which controls the disease, is manufactured only in the *beta* cells of the islands of Langerhans scattered through the pancreas and weighing only one-twentieth that of the entire gland, made the evidence of the unity of diabetes complete.

Subsequently, the close connection of the pituitary gland, situated in its bony case in the center of the skull, with diabetes was recognized, and, later, it was found that actual diabetes could be brought on by injections of an extract of its anterior portion. At first, this reawakened the thought that the nervous system after all was independently involved in the causation of

diabetes and that diabetes was perhaps influenced both by functional as well as organic insults to the brain. Such suppositions, however, were soon dispelled when investigation disclosed that the diabetogenic action of the anterior pituitary extract lay in its power to destroy the cells of the pancreas which produce the insulin and thus caused the disease. Thereby the unity of diabetes and its localization in the pancreas was again demonstrated. (Very recently the influence of the suprarenal gland upon carbohydrate metabolism has received fresh emphasis.) An injection of the extract of its cortex will accentuate diabetes and, conversely, removal of the cortex will lessen its severity. In rats, Ingle has produced a diabetic state by injection of massive doses of cortical extract, but this does not persist when the injections are interrupted. Evidence regarding changes in the pancreas is as yet not available. I know of no instance in a human being in which disease or injury of the adrenal gland has brought on diabetes.

Today, to prove that trauma causes diabetes one must show that the pancreas is gravely injured, in fact, at least four-fifths destroyed, probably nine-tenths, or that the trauma has so acted upon the anterior pituitary gland as to cause it to discharge an excess of extract which, in turn, can destroy the insulin-producing cells of the pancreas. These are the two methods, but examples of the same are almost unknown in humans. For the pancreas, to my knowledge, there exist only the cases of Wells, Stern, and the two of Grafe, which have gained general recognition as proving injury to the pancreas led to diabetes, and not one of these cases is above criticism. As for trauma to the pituitary leading eventually to diabetes, the evidence is still less convincing, and Rathery's recent description of an acromegalic with a complicated head injury, is only remotely conceivable as an example of stimulation of pituitary remnants to excessive secretion. As a matter of fact, he did not attribute the diabetes in this acromegalic, following removal of an eosinophilic adenoma of the pituitary, to the pituitary itself, but rather to the injury of the neighboring hypothalamic region.

Grafe's case of trauma causing diabetes, in my opinion, is the most striking of any in the literature. A condensed report of it is herewith appended:

Grafe's Case.—A merchant, age 75, supposedly free from diabetes, *en route* to a bath cure on account of gallstones, while driving at 50 miles an hour, put on his brakes sharply to avoid a motorcycle and hit a tree. His chest and abdomen were pressed against the steering wheel. No pain followed the accident and he resumed driving, but within three hours developed a gallstone attack. Nevertheless, he continued his journey the next day with a hired chauffeur but, the attack persisting, transferred to a train. Upon arrival at the Spa two days after the accident, he was somnolent, had an acetone breath, showed blood in vomitus and stools and had marked thirst. The glycosuria was 4.7 per cent, blood sugar over 500 mg. Upon entrance to Grafe's Clinic that same day the breath had an acetone odor, glycosuria 4.5 per cent, marked reactions for acetone and diacetic acid, blood sugar 556 mg., no vomiting. There was pallor, pulse good, hemoglobin 85 per cent, abdomen perfectly soft, and although no pain on pressure, cautious palpation revealed indistinctly a sausage-shaped tumor. Glycosuria and acidosis were controlled during the night with 160 units of insulin but

although conscious in the morning with blood sugar 84 mg., he failed rapidly and died at 8:30 A.M. Autopsy showed thrombosis of the splenic vein with pancreatic apoplexy, thrombosis of a branch of the portal vein, fat necrosis of the omentum and mesentery, diffuse peritonitis, and general congestion of the organs.

Grafe's case would appear to show that an injury to the pancreas could be a direct cause of diabetes. However, I have not met with such an instance among the 22258 patients consulting me on account of sugar in the urine. Although his case fulfills many of the criteria essential to explain the onset of diabetes as the result of an accident, nevertheless, its deficiencies are plain: First, diabetes was supposedly absent but not proved absent before the accident. The age of the patient, his sedentary occupation, the association with gallstones, which so often are present in the obese, along with diabetes, raise grave doubts as to the development of diabetes *de novo*. Criticisms of this nature can be raised with nearly every case of so-called traumatic diabetes, but of course are not wholly valid. The case, however, is a model one in these respects because: First, an attempt was made to rule out the presence of diabetes before the accident; second, the accident was of an overwhelming nature to the abdominal region; third, the diabetes followed closely upon it; and, fourth, the autopsy disclosed an extreme injury to the entire pancreas. At the same time, it emphasizes how extraordinarily rare such an incident could occur and the patient live long enough to have diabetes.

Injury to the Pancreas without External Bodily Trauma.—The pancreas being the center around which diabetes revolves, any avenue of attack upon that organ, even indirectly due to trauma, must be considered. There are various possibilities.

Pancreatitis: This is not an uncommon condition. When an inflammation attacks the gland it is generally severe, extensive and serious, and usually results in death. Strange to say, pancreatitis is seldom accompanied by diabetes. I have had one case among my 22,258 diabetics in which it appeared to be of causal significance. Umber, in his series of 7,000 diabetics, reports one instance, and although others less well authenticated have been described, the total number of cases either in a series of diabetics or in a series of cases of pancreatitis is surprisingly small. Nevertheless, if trauma could be shown to produce pancreatitis there would be some ground for considering it as leading to diabetes. The gallbladder is so near the pancreas that the possibility of infections spreading from it into the pancreas and causing diabetes appealed to me for many years. Statistical evidence based upon my own cases and upon studies of the Metropolitan Life Insurance Company forced me to give up this idea.

Hemochromatosis is a disorder of metabolism which leads to a deposition of iron pigment in various cells of the body. Usually it is accompanied by diabetes obviously brought about by deposition of the pigment in the cells of the islands of Langerhans of the pancreas which ultimately leads to their destruction. It was suggested by Mallory that the ingestion of copper was

the cause, but as yet that explanation has not been generally accepted. Conceivably, exposure to poisoning by copper or to anything which would bring about hemochromatosis might be adduced as trauma and thereby an indirect cause of diabetes.

Cancer involves the pancreas and exceptionally is accompanied by diabetes. It is not always easy to say which disease begins first, but there are a few instances in which the author reporting the case considered that the onset of cancer was first and diabetes followed. But if there is evidence that trauma can cause cancer of the pancreas, I do not know of it.

Infections: It is true that diabetes is occasionally discovered at the time of an infection, but infections are so frequent and the detection of diabetes so rare during them that statistical evidence pointing to cause and effect is slight. Priscilla White found in our diabetic children that the incidence of infections in the preceding history was distinctly less than the incidence of infections in children generally. Lande, writing from Umber's clinic, came to the same conclusion.

An infection makes an existing diabetes more severe is a statement commonly acecpted, but there is, likewise, agreement that following the subsidence of the infection the type of the disease returns to its former level. Prolonged infection, whether due to the pyrogenic group of bacteria or to the tubercle bacillus, finally ceases to aggravate the diabetes, as my colleague, H. F. Root, has shown. The fundamental principle that an infection makes the diabetes worse is so generally accepted that it need not be further elaborated here.

Injuries to the Nervous System: Both psychical trauma and organic trauma to the nervous system have often been adduced as a cause of diabetes, but both were dissipated by the World War. Labbé, in France, did not consider trauma an etiologic factor in a single one of 600 diabetic soldiers. I was medical consultant at Mesves hospital centre, through which passed 38,765 soldiers, and there were but three cases of diabetes, two of whom I saw, and there was not the slightest indication that they were related to psychic or organic trauma. "Yet the World War presented an ideal opportunity for the physical and psychic traumatic origin of diabetes both in the combatants and noncombatants and that the disease did not materialize is most significant. The fear of an operation or of the pain incident to child-bearing and the extraction of teeth do not bring on diabetes, neither do we know, nor have we read in the literature, of a surgeon who postponed an operation for fear that the trauma incident to it would cause diabetes."

I know of no instance in which diabetes has been caused by accidents in the course of college athletics, particularly football. Dr. Arlie Bock, Department of Hygiene of Harvard University, wrote me in 1940: "As far as I can determine, no case of diabetes following trauma has occurred among athletes at Harvard. We have had many types of injury, but no known injury of the pancreas, and whether such trauma might result in diabetes I do not

know. You know there has been close medical supervision of athletes at Harvard for at least 25 years." A medical observer of pugilistic contests for 25 years assured me that he had never come across a single person who ever had any symptoms or any knowledge that he had diabetes or had been suffering from diabetes.

Dr. Harvey Cushing reported no instance of diabetes following the development of a tumor in the brain, save those in which diabetes occurred in connection with acromegaly and basophilism, and two patients (out of over 200) with chromophobe adenoma. "What is very significant," according to Dr. Louise Eisenhardt, "is that in Dr. Cushing's own long experience in operating for tumors of the hypophysis or third ventricle he found that such operations did not result in even a transient glycosuria." Dr. Gilbert Horrax, now in charge of the Neurosurgical Department of the Lahey Clinic, formerly Dr. Cushing's associate, confirms what Dr. Eisenhardt has written. Dr. Donald Munro of the Boston City Hospital writes: "I can cite the fact that, in over 3000 craniocerebral injuries, I know of no case in which the trauma had produced either diabetes mellitus or glycosuria by the time the patients had left the hospital after treatment for their injuries." In Germany, Jacobi quotes Liniger as reporting 300 severe head injuries without the development of diabetes and cites Bürger and Poppelreuter to the effect that among several thousand brain injuries no case of definitely traumatic diabetes was observed.

Opinions Pro and Con Trauma as a Direct Cause of Diabetes.—Viggo Thomsen, in his monograph of 416 pages, concludes: "Theoretically, it must be admitted that diabetes can appear as a direct sequel to a pancreas trauma which gives rise to extensive destruction of the pancreas. Other physical traumata are unable to cause diabetes. A physical trauma may give rise to an exacerbation of existing diabetes, but the exacerbation manifesting itself immediately after the trauma is temporary only. Thus the assertion frequently set forth that a physical trauma is able to exacerbate a latent diabetes so that the disease, owing to the trauma, becomes manifest cannot be maintained."

He reaches the above conclusions after an historical, clinical and experimental review of the subject. His conclusions are based, first, upon a study of 144 surgical accident cases admitted to the Aarhus District Hospital, in Denmark, to 100 of whom he gave a glucose tolerance test within three days after the accident. Of the 50 showing disturbances of carbohydrate metabolism, and discharged, he followed the course of 47 for periods of 6 to 38 months. Second, he investigated the effect of the accident on 100 injured diabetics. Third, he compared the antecedent history of trauma in 457 diabetics and a similar number of nondiabetics. "The results of this examination indicate that there is scarcely any difference in the frequency of occurrence of diabetes in injured and noninjured persons of the same age and standard of life." Finally, fourth, he summarizes the details of 81 cases of reported alleged diabetes in the literature.

Lommel, however, is a recent writer who takes up the other side of the question and presents it forcibly, citing the authors who support his view. He argues against rigid dogmatism in so complicated a condition as the causation of diabetes. His theme is that it is not right to assert that because a given explanation *ought not to be* correct is a reason that it *cannot be* the explanation. He assumes the cause of diabetes to be unknown and that it probably comes from an interrelation of many circumstances. He points out the paucity of changes in the pancreas in diabetic children, but he overlooks here, to my mind, the transitory nature of hydropic degeneration when diabetes first appears and the slow development of hyaline changes in the islands in the young. As for heredity, he considers this disposition to diabetes well-nigh universal. He does not believe in a sharp distinction between glycosuria and diabetes and thus is in definite antagonism to men, such as von Noorden, with large clinical experience. To some extent he concedes psychic trauma can lead to diabetes, but to this conclusion I suspect he is evidently somewhat influenced by Strieck's experiment on injury to the hypothalamic area, which has not yet been confirmed. On the other hand he demands (1) that the subject must be proved to be not obviously sick before the accident; (2) that the trauma must be severe, either acting externally with force or exerting a sudden, powerful, psychic provocation; (3) that the glycosuria shall be lasting; and (4) appear in not too remote a period from the accident.

Lommel, in writing his article, has evidently not been in touch with recent experimental work outside of Germany, which has so greatly clarified the problem of the causation of diabetes, and indeed his ideas are also contrary to the prevailing view in Germany itself when he wrote.

Troëll, in Stockholm, in a carefully prepared article, reports ten cases in which glycosuria or diabetes was present with trauma, and even passed upon by the board of reparations. In only two of these was the trauma considered as a cause of the diabetes. The evidence by no means was as strong as in Grafe's case and, in one, followed an injury to the elbow, and the *post hoc propter hoc* argument was raised because the urine was said to be sugar-free the day before the accident. In two instances temporary glycosuria followed a pistol shot wound of the chest or a fracture of the internal malleolus, in a fifth, the later entrance of cancer into the situation led to compensation, in two, trauma delayed, and in two, did not delay healing, and in fact the diabetes was directly responsible *per se*. The tenth, was a case of long duration in which the injury was insignificant and pneumonia decisive. These cases are carefully reported and the circumstances of each are clearly discussed. Troëll, like Lommel, is unwilling to concede that trauma to the pancreas alone can cause diabetes. He believes, as Naunyn emphasized, peripheral injury elsewhere *via* paths of the sympathetic nervous system could be a factor in bringing on the diabetes disagreeing

absolutely with Thomsen. His views seem to me to be biased by Swedish social accident insurance.*

THE ACTIVATION OF LATENT DIABETES BY TRAUMA

The presence of (1) heredity makes an individual susceptible to diabetes, and when to this we add (2) an age in which the incidence of diabetes is high, (3) obesity, (4) reduced exercise, and (5) an infection, the stage is set for a latent diabetes to become manifest. Such a situation is not unusual, and one of the commonest examples is the individual who develops a carbuncle following an almost infinitesimal trauma. With the fulminating carbuncle, the diabetes previously unrecognized, although very likely existent, according to Naunyn, may become critically severe, requiring 100 or more units of insulin, and yet when recovery ensues insulin can be omitted and the diabetes again becomes so submerged that the patient can tolerate more than 200 grams of carbohydrate and, to the unwary, appear cured. But the diabetes is still there, latent, ready to come to the surface again on any provocation. One must extend the importance of trauma far beyond the case of the man with a carbuncle. Any injury to an hereditarily predisposed individual, provided in its train come prevention of customary exercise, exposure to overeating, and particularly if an infection is involved, one must agree, makes a latent diabetes liable to become active. It is by no means necessary that all these factors be present. Proper treatment can control such a diabetes, which is usually mild and may make it appear nonexistent, but the disease once established is really not cured. Space does not allow the presentation of cases, but such have been already published by me. It is the indirect effect of the accident rather than the direct trauma which causes the trouble.

TRAUMA IN THE COURSE OF DIABETES

Trauma is very common in the career of a diabetic. We have 1800 or more diabetics at the New England Deaconess Hospital annually, in ward

* In this connection, a patient is recalled who had been himself an insurance salesman. He maintained in court action, directed against an insurance company, that he had sustained an accidental injury to one toe by stubbing it against a chair on a public excursion boat in Boston Harbor. This injury he claimed had led to infection, and the injury had resulted in diabetes followed by angina pectoris. It happened that he had a peculiar insurance policy which provided for triple indemnity in the event of injury occurring accidentally on a public carrier. If it could have been maintained successfully that this accident had caused not only the injury to the toe, but *via* the sympathetic nervous system, also the diabetes and angina pectoris, under the terms of his contract the total indemnity would have been about $80,000. Actually, in this case the records showed that his diabetes had existed prior to the accident. If such an interpretation as the claimant urged had been accepted by the courts and a precedent thus established, it is easy to see that insurance premium rates would eventually be greatly increased either generally or specifically in patients where diabetes could be established from hereditary family history. Therefore, actually, the acceptance of such a point of view while probably immediately profitable to the one person involved would have social consequences for a large number of people, which would be most costly.

beds and private rooms. At various times, surveys have shown that 45 to 50 per cent of all our diabetic patients, under treatment at any one time in the hospital, came on account of an accident, but this statement is somewhat misleading. Such cases require a longer hospital stay and, therefore, appear proportionately more numerous than they actually are. It would be a fairer statement that one-quarter to one-third of all the patients in the Clinic in the course of a year represented the incidence of trauma. Accidents taking place in the hospital were found to occur relatively six times more frequently among the diabetics as among the nondiabetics. In statistics gathered from New York hospitals the incidence of operations upon the lower extremities in diabetic women was 150 times that in nondiabetic women. The diabetic is more susceptible to trauma than the nondiabetic, because his tissues are more vulnerable.

Purposely, in this discussion I will say little about those controversial features such as tissues of diabetics and the corresponding deficiency in glycogen. Such alterations cannot help affecting the soil, but it is difficult to measure the effect. In one instance we have the normal state and in the other an abnormal status, and that cannot be as advantageous. Then there are the abnormalities due to the products of acidosis; to excess of cholesterol; to an imbalance in the mineral constituents—all, very likely, are important in affecting the resistance of the part but not necessary to prove the diabetic more vulnerable to trauma, because there are so many other features in the diabetic which plainly influence the issue of the trauma for better or worse.

The degree of vulnerability varies with the age of the individual and his circulation, the nerve supply of the part, the severity of the diabetes, the duration of the disease, and its degree of control, not alone at the instant of the trauma, but from the day of onset of the diabetes.

Diabetics at present, based on mortality tables, are living about 14 years and approximately one-half die of arteriosclerosis in one or another of its various manifestations. Add to the developing arteriosclerosis in a diabetic the hardening of the arteries which comes to everyone as age advances, and his susceptibility to trauma increases. Arteriosclerosis implies deficient circulation, and circulation is all important in the healing of wounds. Step on the toes of a child or young adult and he forgets it in a few minutes, but the same blow on the foot of an old man may cause him to be confined to his bed for weeks, and, if a diabetic with poor circulation, he can lose his toe, his foot, his leg or even his life. The blow in one instance is on tissue with good circulation, in the other with poor circulation. On one side of the highway between Phoenix and Tucson the crops are flourishing, but on the other the land is barren and the irrigation canals, the circulation in the soil, show the reason why.

The normally functioning nerve supply of a tissue not only affects susceptibility to trauma, but the ability to heal the injured part. This is easily

overlooked. The wires are there, but they do not always transmit the messages or only a part of them. The diabetic may lose the sense of distinguishing between heat and cold and as with two of my patients the foot gets frost-bitten on Cape Cod or burned in Canada. His sense of pain is impaired and four of my patients have walked around at work all day and on removing their shoes at night found a tack which had evidently penetrated the sole of their foot hours before. My colleague, Howard F. Root, has collected 15 of our cases in which the ankle bones were actually destroyed and without the patients experiencing pain—a condition known as a neuropathic joint, akin to the Charcot joint of syphilis. I have seen a surgeon repeatedly open with strong scissors, in the ward before a group of visitors, nonnecrotic areas in diabetic feet for drainage or other reasons, making cuts one or two inches long without any anesthetic or any sign of discomfort on the face of the patient. It is seldom we do not have a patient with anesthetic feet in the hospital, and so vicarious is the absence of sensation that one foot may be anesthetic and the other not. I suspect that some of our patients who have gangrene originating from trivial exposures may have their lesions dependent upon lack of trophic nerve activity. Then there is the liability to accident because of loss of power due to the nonfunctioning of the motor fibers of the nerve. Common examples are toe-drop (peroneal paralysis), inability to rise from a chair (quadriceps paralysis) and double vision (external rectus), and, in rare instances, almost complete paralysis of the muscles of the entire body. Is it any wonder such people have accidents? The neuritis may affect the nerves of the intestines, resulting in an almost intractable diarrhea or those of the urinary bladder with distension and paralysis of that organ. In Walla Walla, Washington, just at the edge of the city, there are fields of luxuriantly growing onions and I was told the land was worth $1000 an acre, but across the way the property looked the same to me, but was uncultivated simply because a mineral was lacking in the soil. Some diabetics are lacking in an efficient nerve supply.

The severity of the diabetes affects the vulnerability of the diabetic. His disease may be so mild that it is almost impossible to distinguish between health and diabetes, and this status may last for 10–20 years if moderate discretion in living is observed. Contrast this type with that of the severe diabetic who with exposure to extra exertion, directly incident to trauma, can go into coma. The liver of a mild diabetic is good enough to allow him to take ether, but give it to a severe diabetic, uncontrolled for the time being, and he dies of acidosis, just as Case No. 729 did, who took ether for a dental extraction on Tuesday and died on the following Friday of diabetic coma in preinsulin days. The severe diabetic is the one who has the complications affecting the circulatory and nervous systems, complications of the eyes with all that goes with impairment or loss of sight, and even complications of the bones in part due to their decalcification. This is manifest even in young patients. A review by Eisele of our 73 children, onset

under 15 years of age, who have survived 20 or more years of diabetes, illustrates the seriousness and frequency of complications in the diabetic which make him a prey to accidents. Thirty per cent of those examined roentgenologically showed peripheral arteriosclerosis, 55 per cent moderate to advanced arteriosclerosis, and 70 per cent of the patients showing these changes had either a high blood pressure or albuminuria, or both, at an average age of 29 years.

An infection makes a diabetes worse. To that all agree. The instant an infection enters the picture of trauma a whole train of disastrous consequences is set loose. The infection not only makes the diabetes more severe, but it usually prevents exercise, a most desirable adjunct to diet and insulin in the triology of treatment, confines the patient to bed, with the possibility of contracting pneumonia, bed sores, and, with the old, to disarrangement of life-long habits of regulating the action of the bowels and urinary tract, which in the end may prove to be far more serious than the accident itself.

The duration of the disease counts. How can it help it? Before the discovery of insulin, up to 1914, my patients lived 4.9 years, and 1.8 per cent survived 20 years. Today, fortunately, the average duration has lengthened to 14.3 years, and 19.1 per cent survive 20 years, and eight per cent over 25 years. They cease to live because they are more vulnerable. Their life expectancy was two-thirds, and I hope in the next series studied will be three-fourths that of their nondiabetic confrères. Although not always measurable, yet with each added year of a diabetic's life there is some impairment in bodily function.

Finally, the degree of control of the disease counts too. Insulin has proved that for the average diabetic. Even before insulin was discovered adherence to the diet and good hygiene enabled many a moderately severe diabetic in the older age-groups to live. Between 1922 and 1929 gangrene caused 8.5 per cent of our 1448 deaths, but between 1937 and 1940 only 4.7 per cent of 929 deaths. The duration of the disease was 8.1 years in the former period but was 12.5 years in the latter, the average age at death being, respectively, about 63 and 67 years. Thus, better control of the disease despite its longer duration and the older age of the patients reduced the mortality from gangrene which in about half our cases was provoked by trauma. The same degree of trauma can play havoc with a poorly controlled diabetic which would be tolerated with comparatively little trouble by a diabetic living up to the rules. Most of the difficulties of the diabetic are of his own choosing.

TRAUMA IN CONNECTION WITH THE USE OF INSULIN

Within a very few months after the use of insulin in human beings instances of infection at the site of injection of insulin ceased to occur. Among 1838 admissions to the George F. Baker Clinic during 1941 there were but 8 who entered for abscesses due to the injection of insulin. When one considers that only this small number of incidents occurred among many million injections in patients both inside and outside the hospital, it is evi-

dent both the manufacturers and the patients use care. Needles broken in the skin during injection have never led to serious trouble in my experience, and such occurrences are even more rare than abscesses. A far more frequent, and infinitely more serious, opportunity for trauma is that incident to an insulin reaction. However, despite the thousands of insulin reactions occurring in the course of treatment of diabetics, I do not remember one resulting in a fracture. This is in marked contrast to the incidence of fractures when insulin is employed to produce convulsions in nondiabetic neurologic patients. I did observe one case in which death appeared to result from the patient, while unconscious, having regurgitated food which plugged the trachea. Still more serious are those cases in which an insulin reaction has been mistaken for diabetic coma and, in consequence, a dose of insulin has been given which resulted in death. Fortunately, such instances are few, in fact, I know of but nine among 15,000 or more of my patients who have had insulin administered. Four of these received additional insulin while in shock, one, undoubtedly, took a lethal dose of insulin with design, and the circumstances regarding the remainder were somewhat obscure. None of the cases were observed in Boston, and but five were seen in consultation.

In the preparation of this article I have been aided by too many friends to mention here, but I am especially indebted to Dr. Howard F. Root, my colleague of 24 years, who not only has given detailed help but agrees with me in the views expressed.

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RELATION OF TRAUMA TO SYPHILIS OF THE NERVOUS SYSTEM

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In the light of our present knowledge of the pathology it may seem a little strange to discuss the rôle of trauma in the causation of syphilis of the nervous system. In the days before the discovery of the Wassermann reaction and the demonstration of spirochaetes in the brain and spinal cord of patients who had died of neurosyphilis, such a discussion would seem less out of place. Now that it has been proven that the signs and symptoms of neurosyphilis are due to the reaction of the body to the invading organism, the spirochaete, it would seem just as logical to discuss the rôle of trauma in the causation of scarlet fever or typhoid fever except for the fact that in most cases of neurosyphilis the onset of the signs and symptoms of involvement of the nervous system by the spirochaetes, may not appear for several or many years after the initial infection. During this interim the individual may be subjected to various influences, including trauma, which may be considered in certain instances to play a factor in the onset or progress of the symptoms. It is the purpose of this article to consider to what extent trauma can be considered of importance in the production or exaggeration of symptoms in the various types of neurosyphilis. It must be emphasized that in no case can trauma be considered as the sole or main cause of the signs or symptoms of syphilis of the nervous system. Our consideration must be limited to the evaluation of trauma as a secondary factor; whether trauma may cause changes in the nervous tissues which may make them more vulnerable to invasion by the spirochaete, or produce changes which will increase the number of symptoms or the progress of the disease in patients whose nervous system has already been invaded by these organisms. In addition to the severity of the injury, the time interval between the injury and the onset of symptoms is an important factor to be considered.

For clinical purposes cases of syphilis of the nervous system may be subdivided into three groups: 1 Meningeal, 2. Vascular. 3. Parenchymatous.

MENINGEAL NEUROSYPHILIS

In meningeal neurosyphilis the inflammatory process is confined to the coverings of the brain and the symptoms are due to irritation of these coverings and disturbance of function of the nerves which traverse them on the way to their exit from the skull or spinal canal. Thus, there are such symptoms as headache, nausea and vomiting together with disturbance of the function of one or more of the cranial nerves (paralysis of one-half

of the face, unilateral deafness, or paralysis of one or more of the muscles moving the eyeballs). Involvement of the meninges by syphilis usually occurs early in the course of the disease and it is common for the symptoms to have an acute onset within a few months of the appearance of the primary lesion. Occasionally the onset of symptoms may be delayed for many years after the infection. Since the symptoms may have a rather dramatic onset in this type of neurosyphilis, it is not strange that in an occasional patient such symptoms may appear to have a temporal relationship to some injury of a severe or trivial nature. It may be quite difficult in such cases to prove whether the injury has any relationship to the onset of the symptoms of meningitis. Several cases are reported in the literature in which the symptoms had their onset after a head injury. In a study of 80 cases of acute syphilitic meningitis which was made in 1935, we were unable to find any such coincidence. Since this report, however, we have seen two cases in which the onset of symptoms followed a head injury.

The first patient, a white male, age 27, was admitted to the hospital with complaint of severe right-sided headache and vomiting without nausea of five days' duration. Two days prior to the onset of symptoms he had been in a fight and had received a kick in the head but had not been rendered unconscious. There was a history of a penile chancre and a secondary skin rash six months previously. The neurologic examination was normal except for stiffness of the neck. The cerebrospinal fluid contained an excess of white blood cells and the Wassermann reaction was positive. The patient recovered rapidly with antisyphilitic treatment.

The second case was a white male, age 42, who was in an automobile accident in January, 1937. He was unconscious for an unknown interval and suffered a fracture of the leg. He was hospitalized for 11 weeks, during which time the cerebrospinal fluid was not examined. After discharge from the hospital he began to have severe headaches. These headaches continued and, in September, 1937, he began to have nausea and vomiting. He was readmitted to the hospital for study. The blood serologic tests were positive and the cerebrospinal fluid contained an excess of cells, and the Wassermann reaction was positive. The symptoms were rapidly relieved by antisyphilitic treatment.

In analyzing these two cases it would seem unlikely that the minor degree of trauma suffered by the individual in the first case cited above could have much relationship to the meningeal symptoms, since the time interval between the injury and the onset of the full-blown clinical picture was too short for the injury to have had any significant influence on the pathologic process. The situation is somewhat different in the second case where the injury was more severe and the interval between injury and appearance was such that it makes more tenable the hypothesis that the traumatic injury to the brain and meninges made these structures more vulnerable to the syphilitic infection.

In summary then, it may be stated that the incidence of syphilitic meningitis following head injury is an extremely rare occurrence. The burden of proof is, therefore, on the individual who tries to demonstrate such a relationship in any given case. Syphilitic meningitis is a relatively rare

clinical entity and, as stated above, usually has its onset in the first few months after the primary infection. An ideal case to prove the relationship of head injury to the development of syphilitic meningitis would have the following: 1. Primary infection several months before incurring a severe head injury. 2. Spinal fluid removed immediately after the injury showing no excess of white blood cells and negative serologic tests. 3. The development of symptoms one to eight months after the injury. 4. Presence of an excess number of cells and positive serologic tests after such symptoms have developed. It is perhaps too much to ask for the complete fulfillment of all of these criteria and each case will have to be considered on its merits with particular regard to the number of criteria which it fulfills.

VASCULAR NEUROSYPHILIS

Involvement of the vessels of the nervous system by syphilis produce signs and symptoms similar to those developing in patients with disease of these vessels due to other causes, such as arteriosclerosis. Since the pathology of neurosyphilis is chiefiy one causing a thickening of the vessels the mode of production of symptoms is more commonly due to an occlusion (thrombosis) of these vessels than a rupture. The signs and symptoms produced depend upon the location and size of the vessel involved. Such signs as hemiplegia, aphasia, hemianopia, *etc.*, are most commonly seen. In general, signs and symptoms of vascular neurosyphilis usually occur at two different stages of the infection. In the early stages of the disease, they may occur along with signs and symptoms indicating involvement of the meninges (syphilitic meningitis). At this time the vascular pathology is an occlusion of the vessels as a result of the inflammatory reaction in its outer sheath. In the later stages of the disease, proliferative changes (endarteritis) in the vessel lead to an occlusion. In only a few of such cases can the structural changes in the vessel be pathologically proven to be due to syphilis. In many cases the changes in the vessels are of such a nature that the pathologist is unable to state definitely the etiologic factors. This is particularly true when the affected individual is an elderly or middle-aged patient and also suffers from arteriosclerosis of a minor or severe degree.

The proof of the relationship of trauma to early vascular neurosyphilis is the same as that for the proof of syphilitic meningitis. In late vascular neurosyphilis the criteria for proof cannot be so definitely stated and should be the same as those for vascular lesions of any cause. It is commonly accepted that a severe injury to the head may be the cause of rupture or occlusion of vessels in patients with normal vessels and these are much more apt to happen in patients with arteriosclerotic vessels. It is logical to assume that such a trauma may have a similar effect on vessels damaged by syphilis.

The following case is cited to illustrate the occurrence of symptoms after a head injury in a patient with presumed vascular neurosyphilis:

A colored female, age 37, was hit on the head by a baseball bat August 23, 1932. The skull was fractured and there was an injury to the right seventh nerve, with

complete paralysis of the fascial muscles on this side. Eighteen days later the patient fell to the floor unconscious while working in the kitchen. On recovery of consciousness there was a complete paralysis of the left arm and leg. The blood pressure was normal. Serologic tests of the blood were positive and there was an increase in the number of white cells and protein content of the cerebrospinal fluid.

In summary, it can be concluded that since trauma to the nervous system may damage normal vessels it is more likely to damage vessels already diseased by syphilis. The proof of causal relationship must rest upon an evaluation of the severity of the trauma and the interval between the trauma and onset of symptoms. Careful consideration must be given to the events connected with the presumed injury, since it may be possible that the onset of the vascular accident and resulting unconsciousness may be the cause of the so-called injury. For proof of causation it must be shown that: 1. The injury was *bona fide* and severe enough to damage nervous tissue. 2. That the symptoms had their onset immediately or within a reasonable interval after the injury.

PARENCHYMATOUS NEUROSYPHILIS

Involvement of the parenchyma of the brain usually takes the form of two well recognized syndromes: 1. Paretic neurosyphilis. 2. Tabetic neurosyphilis.

(1) *Paretic Neurosyphilis.*—Paretic nerosyphilis (dementia paralytica) is characterized pathologically by a diffuse encephalitis which is most intense in the frontal lobes of the brain. The clinical picture may be quite varied but personality changes and mental deterioration are prominent early symptoms. Convulsive seizures or transient neurologic signs, such as hemiplegia or aphasia, as well as any type of psychotic manifestation may be the initial sign of the disease. The interval between primary infection and development of symptoms is usually 10–20 years. The literature of 50 years ago contains considerable discussion as to the rôle of trauma in the production of paretic neurosyphilis but very little significance is given to this factor in modern literature, probably because the understanding of the disease makes the hypothesis of injury unnecessary. Pearce Bailey, in his classical treatise on Diseases of the Nervous System Resulting from Accident and Injury, in 1906, cites eight articles on the relationship of injury to paretic neurosyphilis. The authors cited by him gave the incidence of head injury in these patients as varying from 4.4 to 43 per cent. Bailey, himself, was skeptical of these reports and points out the difficulties in evaluating the rôle of trauma in the production of neurosyphilis, and states that in many cases the injury was a direct result of the disease itself (falls in convulsive or apoplectiform seizures or blows received in brawls precipitated by the psychotic behaviour of the individual), or the interval between the injury and the onset of symptoms was such that a causal relationship could not be considered tenable. The modern viewpoint, as expressed by such authors as S. A. K. Wilson and Kraepelin, tends to the view that if trauma

plays any rôle, it is quite a subsidiary one. Wilson states that the supposition is incapable of rigid proof that injury can evoke a morbid activity on the part of the treponeme which otherwise would not have been evidenced or that it can light up a latent process or intensify one already existent. He also states that there was no evidence of any such factors operating as result of head injuries in the first world war. Kraepelin states that in our present state of knowledge it is not possible to prove or disprove that an injury to the head has an unfavorable influence on a case of paretic neurosyphilis. Klauder and Solomon reviewed the subject in 1931, and it was stated: "That any conclusion as to the relation of cranial injury to a subsequently appearing dementia paralytica should not be reached solely on the length of time between the injury and appearance of the disease, but rather by correlation of the history antedating the injury; the nature of the injury; the character of the posttraumatic symptoms and their relation to the appearance of diagnostic symptoms of dementia paralytica."

The citation of several case histories where the development of symptoms followed a severe head injury may be of value:

Case 1.—A workman, who according to all accounts, was in perfectly normal physical and mental health, was blown through a double window in an explosion, badly bruising his head. A few weeks after the accident the patient began to show signs of mental disorder, performing foolish acts, showing evidences of memory loss, and finally becoming entirely unfit for work. Eight months later, examination in the hospital showed that the patient was confused, disoriented, and that he was given to foolish laughter. His judgment was greatly impaired and his memory was poor. There were signs of central nervous system disease, as indicated by reflex changes. The blood and spinal fluid examinations were typical of general paresis, which diagnosis was made.

Case 2.—A male, age 40, in apparently normal health, both physical and mental, was skating with a party of friends when he fell on the ice, striking his head a severe blow. He was rendered unconscious immediately. He was brought 60 miles to a hospital, and it was only after many hours that he began to regain consciousness. The clinical diagnosis was concussion with probability of subdural hematoma. A lumbar puncture was done which showed relatively little increase in pressure. While the matter of craniotomy was being discussed, examination of the spinal fluid gave the characteristic findings of general paresis. The patient never regained his normal mentality, requiring hospitalization in a mental institution, and despite antiluetic treatment has run the usual course of a paretic.

The judicial point of view can best be presented by abstracts from a Connecticut case designated in the legal parlance as "Barry vs. Miller, *et al.*, 104 Conn. 462; 133 Atl. 37 (1926)."

This was an appeal by the defendants from a finding and award of compensation in favor of the plaintiff. The appeal was dismissed.

Case 3.—The plaintiff, while working as a plasterer, stood upon a staging which fell, causing him to be thrown to the floor, a distance of about four feet, and to strike the floor in a sitting position. He was very pale, his eyes were closed, and he remained unconscious for from five to ten minutes, and upon regaining consciousness complained of pain in his back. He continued at work for about a month, complaining constantly of pain in his back, and in about ten days after the injury began to show

signs of nervous irritation and mental weakness. His nervous system and mentality were apparently normal before the injury. One and one-half months after the injury he was admitted to a hospital for the insane. While there he was found to be suffering from general paresis and that he had been suffering from syphilis many months prior to his fall.

The commissioner who heard the case found in Paragraph 13 that: "The injury of November 8, 1923, lighted up the syphilitic condition, and either caused or accelerated the onset of the paresis." The ground of appeal of the defendant is that this finding is made without evidence.

The Court, in its opinion, states as follows: "The evidence developed these further facts: The claimant had been, until the accident of November 8, in good health, physical as well as mental, and apparently with none of the objective or subjective symptoms of the disease paresis. The fall injured claimant's head slightly, as well as his back. When taken to the hospital on December 15, he was found to be suffering from acute paresis in an advanced stage caused by syphilis, it having existed for at least several months and causing him to become insane. Paresis is a chronic progressive disease. No cause for the onset of this acute form of paresis with which the claimant suffered following the fall was suggested by the two medical experts for the defendants, and none appears in the evidence unless the fall occasioned the onset.

"The experts for the defendants testified that this onset of paresis bore no relation to the fall, but they gave the commissioner no basis for this opinion other than that paresis was a germ disease and could not be caused by an injury either to the back or the head. The expert for the claimant testified that a latent disease such as paresis may be precipitated by an injury and that the injury which the claimant had suffered was sufficient to have precipitated this onset of paresis. This expert testified that he could not state positively that the injury precipitated this onset of paresis, but that it might have been accelerated by the fall or any shock to the nervous system; that if the claimant had not symptoms of paresis prior to the fall, and the symptoms of paresis developed immediately afterward—and that was true in this case—it was an exciting cause. This opinion he qualified by saying it might have been an exciting cause. In response to the commissioner's question, 'What are the probabilities or aren't there any?' he replied, 'I think that the probabilities are that anybody suffering with a disease, any kind of a chronic disease, chronic kidney trouble, or chronic lung trouble, any sudden shock to the nervous system, or shock to the body, especially with some disease of the nervous system, a sudden shock to the body would be apt to precipitate the disease, make it break out quickly.' The opinion of this expert, who testified as defendant's two experts had testified, was not later attacked or questioned by either of them.

"In this condition of the evidence, we cannot hold that the commissioner erred in finding that the injury precipitated the onset of the paresis. The commissioner might reasonably have found that the injury not only might have been, but that it probably was the occasion of this onset. The onset of a chronically progressive disease came upon the claimant, a man apparently well, physically and mentally, without a symptom or warning of its presence. There was no other cause suggested in the evidence. It was not only a possible but a reasonably probable cause. If the commissioner concluded from his evidence that the injury was a reasonably probable cause of the onset, it was his duty to find as a fact, as he has found that it did cause or accelerate the onset of the paresis."

From these records there is medical evidence and judicial precedent to connect trauma to the head with the development or acceleration of the paretic process. From a pathologic viewpoint it is important that a suitable interval elapse between the injury and the development of symptoms. In

the cases cited above, the injury could be sufficiently severe to cause damage to the nervous system and in the first and third cases the interval between the injury and the appearance of symptoms was sufficiently long for any deleterious effect of the injury to have influenced the paretic process. In the second case, the interval appears too short for such an effect. Similarly, appearance of symptoms after an interval of several years would hardly be appropriate. In general, the limits set by most authorities for a causal relationship between head trauma and the development of paretic symptoms as a minimum is six weeks to three months and as a maximum, two to three years. It is also logical to assume from the medical viewpoint that injuries to other parts of the body than the head can have no causal relationship to the development of paretic neurosyphilis nor can such factors as emotional shock be considered of any importance.

(2) *Tabetic Neurosyphilis.*—The situation with regard to tabetic neurosyphilis is analogous to that of paretic neurosyphilis, as discussed above. Pathologically, the two conditions are similar in that the primary involvement is in the parenchymatous tissues of the nervous system and there is a long latent period between the initial infection and the appearance of symptoms. The main difference between the two conditions is that in paretic neurosyphilis the chief changes are in the cerebral cortex, whereas in tabetic neurosyphilis they are in the spinal cord and midbrain. The symptoms of tabetic neurosyphilis are quite varied but the most common are: Lancinating pain in the body and extremities, disturbance of gait, loss of control of the urinary bladder, double vision from palsy of the eye muscles, atrophy of the optic nerves, visceral pains, trophic ulcers and degenerative changes in the joints.

The problem here, as in all cases of neurosyphilis, is one of precipitation or exaggeration of symptoms. Logically, it would seem necessary that the injury involve the spine to be a factor in evoking the spinal symptoms or to the head for the midbrain symptoms of tabetic neurosyphilis.

A case illustrating the development of symptoms of tabetic neurosyphilis after an injury can be cited:

A vigorous male, age 63, who claimed to have been perfectly well and enjoying the best of health, while playing a game of polo received an injury to his head which did not unseat him from his horse but caused him to feel rather badly. A swelling developed on the back of his head from the blow and he suffered pain in the back of the head and neck. There was no loss of consciousness. He complained subsequently of headache and dizziness. The next morning he got up and went out of the house but felt dizzy and had so much headache he stayed at home most of the day, although not requiring bed care. The following day he developed a diplopia which did not improve and which was present three and one-half years later. Examination at that time showed weakness of both external recti, absent knee jerks and positive serology. The case came to legal action, the plaintiff claiming that although the patient had tabes, the disabling symptoms were the result of a blow he received. Expert opinion differed. The defendant's neurologist stated that a blow that did not lead to unconsciousness would not start up a dormant syphilitic condition and make this condition active. He

further stated that the effect of such a blow would be the same whether the victim were syphilitic or nonsyphilitic. A second expert testified for the defendant that: "It is possible but not probable that a dormant syphilitic condition can be excited into action by a blow on the head that does not result in unconsciousness or fracture of the skull." He recognizes, however, that many authorities hold the opinion that such a result is probable, even though there be no fracture of the skull and no lacerations of the scalp, there may yet occur multiple hemorrhages in the deeper structures of the brain. It was this expert's opinion, however, that, taking into consideration the lack of severity of the blow, the symptoms which occurred were due solely to syphilis and developed entirely independently of any blow.

The expert testifying for the plaintiff held the opinion that a man, age 63, who had no active signs of tabes, would probably not develop such signs unless there was some exciting cause, such as a blow on the head, and that this need not be of great severity to cause the symptoms.

The decision rendered by the court was quite revealing. It was found that medical science could not determine with certainty the results which may come to any one from an injury to his head, and in such cases the decisions and opinions of physicians are not a matter of absolute certainty but are a matter of delicate clinical judgment based upon experience. It was decided that the symptoms, as described, could be explained adequately on the basis of syphilis alone, but the vital point in the given case is whether syphilis alone was responsible or whether the blow caused the development of the disease which otherwise would have given the patient no trouble. The final decision was that the blow was the immediate precipitating cause of the symptoms and the individual, therefore, was compensible.

The relation of the trauma and syphilitic arthropathies, or Charcot joints, affords less opportunity for a divergence of opinion. It is rather generally accepted today that Charcot joints develop as the result of lessened sensitivity in the joints which allow repeated minor traumata to occur without adequate protection by the muscles or tendons. In other words, the usual Charcot joint is traumatic, in the final analysis, in a patient with a defective sensory system. Numerous cases have been placed in the literature showing that trauma was the immediate cause of the development of the syphilitic arthropathy.

The defense of the hypothesis that external trauma can have an unfavorable influence on the tabetic process must be along the lines given above for paretic neurosyphilis. Direct trauma to the spinal cord by the injection of foreign substances into the spinal subarachnoid space (such as spinal anesthesia) while an extremely rare occurrence, may have a deleterious effect on the tabetic process, as illustrated by the following case, which also emphasizes the importance of an adequate examination of every patient before the administration of drugs intraspinously:

A middle-aged white male, who had been previously in good health, was admitted to the hospital for the excision of external hemorrhoids. The records do not indicate that any neurologic examination or serologic tests were made before the administration of spinal anesthesia for the performance of the operation. After the operation the patient was unable to void his urine and there was weakness of the lower extremities. Neurologic examination showed the typical findings of tabetic neurosyphilis, and the cerebrospinal fluid, examined on numerous occasions, contained an excess of cells, an

abnormal colloidal gold test, and the serologic tests were positive in the blood and cerebrospinal fluid. There was gradual improvement of the patient's symptoms during the course of the next few years, during which time he received antisyphilitic treatment.

SUMMARY

The rôle of trauma in the precipitation or aggravation of the symptoms of neurosyphilis has not been adequately established on a scientific basis. The one proven fact is that neurosyphilis is due to the invasion of the nervous system by the spirochaete. The deleterious effect of trauma on the pathologic processes of neurosyphilis can only be deduced. There is divergence of opinion as to severity of injury necessary, and the time interval which must elapse between the injury and the appearance of symptoms. Since there is basis for controversy in individual cases, it is the duty of the medical expert to keep within the limits of what is reasonable and not give the dignity of probability to conclusions that may only be considered as remotely possible. Likewise, it behooves the judiciary, granting that where there is reasonable doubt, the benefit of doubt should accrue to the victim, not to go too far in relating injuries to other parts of the body than the nervous system to the development of neurosyphilis.

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READING LIST OF ENTIRE GROUP OF MEDICOLEGAL ARTICLES APPEARING CONCURRENTLY IN MEDICAL AND LEGAL JOURNALS UNDER EDITORSHIP OF DR. HUBERT W. SMITH

EDITOR'S NOTE: *The articles in this* ANNALS OF SURGERY *symposium are part of a series on Medicolegal subjects appearing simultaneously in many other medical and legal journals. The series is sponsored by Harvard Medical School Department of Legal Medicine and is under the editorship of Dr. Hubert W. Smith, of Harvard.*

JOURNALS

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*2. Annals of Internal Medicine (Ann. Int. Med.); Green and Redwood Streets, Baltimore, Maryland.
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* Devoting entire April 1943 issue to the Symposium.

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BRIEF COMMUNICATIONS

HYDROMETROCOLPOS IN INFANCY

CASE REPORT

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CONGENITAL ATRESIA of the vagina, when coexistent with abnormal activity of uterine and cervical glands, results in distension of the uterus and vagina proximal to the point of obstruction. Mahoney and Chamberlain[1] have recently called attention to the importance of this condition, hydrometrocolpos, as the cause of palpable abdominal tumors in female infants, and have stressed the need for recognition of the complex before unnecessary and dangerous abdominal surgery is undertaken. They have pointed out that absolute diagnosis can be obtained in a suspected case through roentgenoscopic studies after the injection of radiopaque substances through the point of atresia. The logical treatment, incision through the imperforate membrane, resulted in complete regression of signs and symptoms in the single case where a correct diagnosis was made preoperatively.

We have recently had the opportunity to study a classic example of hydrometrocolpos which afforded confirmation of the value of roentgenoscopy in establishing a correct diagnosis and illustrated the importance of acute urinary retention as a complicating factor.

Case Report.—Hosp. No. 127642: The infant, a female, age 17 days, had progressed well for the first few days following an uneventful birth. A progressive increase in the size of the abdomen was noted thereafter, and for several days prior to admission the output of urine had been reduced. The baby screamed constantly as if in pain. Examination showed her to be an adequate newborn. Her abdomen was markedly distended by a mass which extended from under the pubis to a point within three centimeters of the xiphoid. It was rounded, smooth, flat to percussion, and it did not present in the flanks. Presenting at the vagina was a smooth cystic mass which protruded when the baby cried but was not under the same tension as was the abdominal mass. Catheterization yielded 360 cc. of cloudy urine, and resulted in disappearance of the prominent abdominal mass, but revealed a second tumor in the midline, which was the size of a small orange, and, when felt through the rectum, was found to extend into the pelvis and to be confluent with the protuberance present at the vaginal outlet.

Procedure.—A needle was passed into the vaginal portion of the tumor and 30 cc. of mucinous grey fluid was removed and replaced by 20 cc. of radiopaque media. Roentgenograms then demonstrated the tumor to consist of a greatly dilated vagina and uterus (Fig. 1). A cruciform incision was placed through the vaginal protuberance and six ounces of fluid, similar to that aspirated, was allowed to escape, with almost immediate disappearance of the mass. A small amount of fluid continued to leak from the incision for a few hours and thereafter no discharge of any description was evident.

The postoperative course was complicated by pyuria and other evidences of an atonic bladder. Improvement was gradual over a period of two weeks, during which time frequent catheterizations were done and adequate chemotherapy was given. When seen at the age of three months the urine was clear and there was no bladder residual. The point of incision could not be made out at the introitus but there appeared to be a dilated hymenal ring which was free from tabs or other irregularities.

Whether this case is representative of an imperforate hymen or of actual atresia of the vagina has not been answered. Certainly, the protuberance appeared to descend from well above the usual site of the hymen and its wall was rather thick and vascular; suggesting more of an atresia. The fluid obtained by needling was similar in appearance to that so frequently noted in the normal newlyborn female. It was viscid, free from cellular elements, and contained 1.6 grams per cent of protein.

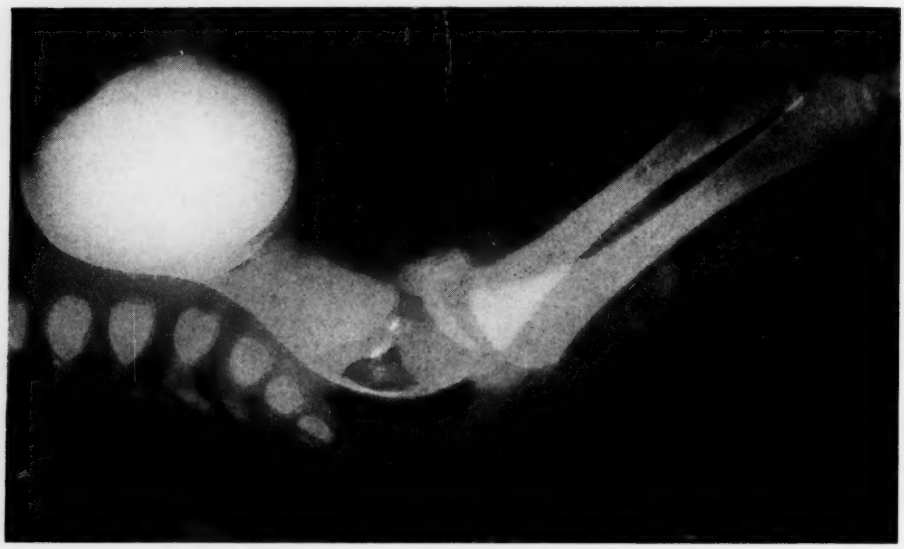

FIG. 1.—Roentgenogram following replacement of contents of tumor with radiopaque media.

SUMMARY

A case of hydrometrocolpos, occurring in a newlyborn girl and complicated by acute urinary retention, is described. Such cases appear to depend upon the concomitant existence of an atresia of the vagina and an excessive secretion of cervical and uterine glands. A correct diagnosis can be established by visualization of the tumor after injection with radiopaque substance, and treatment is confined to incision through the atretic membrane. The true nature of the condition must be recognized, otherwise dangerous, crippling, and unnecessary abdominal surgery may be inadvertently undertaken.

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AN IMPROVED TOWEL CLIP

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A TOWEL CLIP designed to meet the defects of the usual clips has been used with satisfaction for the past year at the Peter Bent Brigham Hospital. There are two defects in the functional design of the ordinary towel

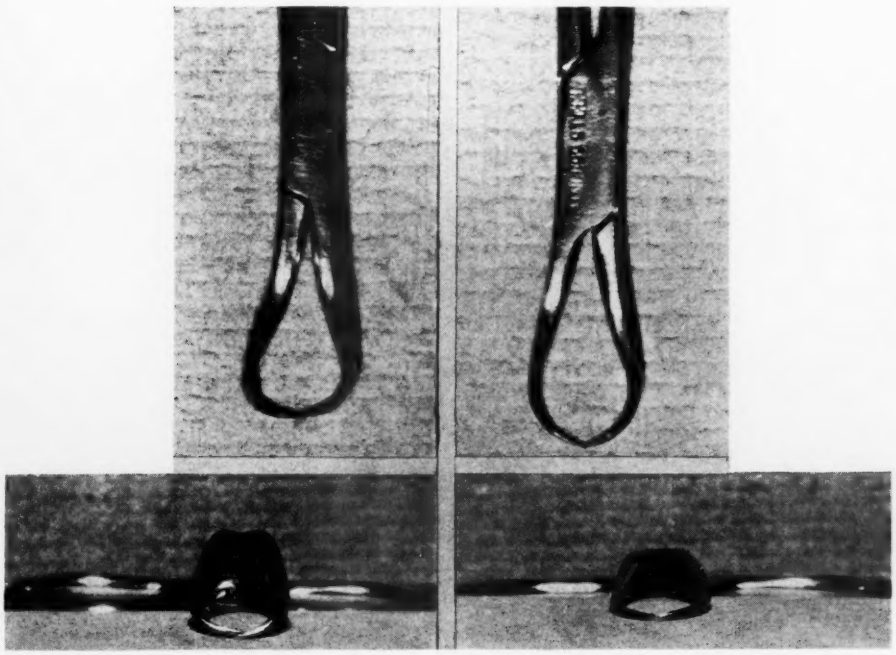

FIG. 1.—The points of the usual towel clip (left) are in the same relation to the surface in all positions. The points of the improved clip (right) point into the skin when it is held at right angles to the surface. When the clip rests on the skin below, the points are tangential, as in the older clip.

clip: First, it is difficult, or impossible, to make the points take a small bite into the skin through a towel, especially if the skin is on stretch, as in the scalp. This is because the points are essentially parallel to the skin surface in all positions of the clip, and the skin, therefore, must be "pinched" into the points before it is perforated. The second defect is that the slope or rise from the tips of the instrument to the box is so marked that the towel clip, in place at the edge of the incision, may interfere with other instruments used in the wound.

The improved towel clip is designed to facilitate the entrance of the points into the skin. A double curve has been placed in the jaws so that, when the clip is held with the long axis nearly perpendicular to the skin,

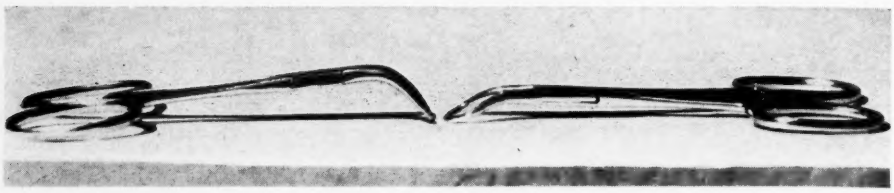

FIG. 2.—The slope of the new clip (right) has been reduced to minimize interference.

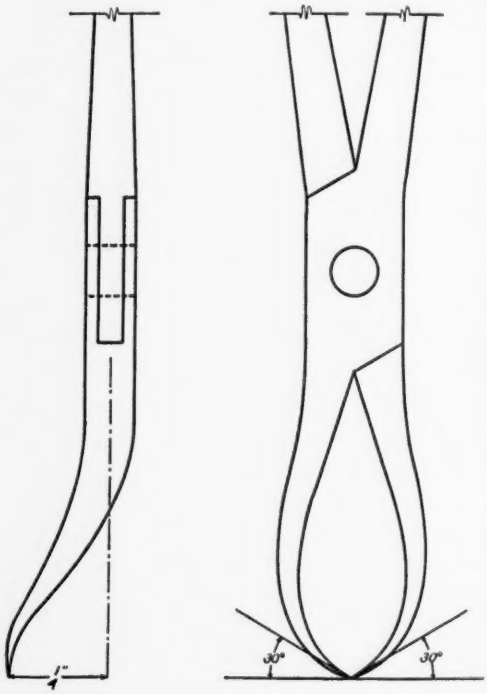

FIG. 3.—Working design of the improved clip.

the points angle down and sink easily into the skin when the clip is closed; but when the clip rests in position on the skin, the points do not angle down into the skin. Figure 1 compares the angle of the tips of an ordinary towel clip and of the improved clip in these two positions. To meet the second defect, the slope of the blades has been reduced so that when the clip is in position on the skin it offers minimum interference. Figure 2 compares the slope of the blades in the ordinary clip and in the improved clip.

Figure 3 gives the dimensions for the manufacture of this type of clip,* which may be obtained in three and one-half- and five and one-half-inch lengths. We prefer the shorter.

The use of this towel clip has permitted accurate and easy fixation of the towel corners to the skin at the line of incision and minimum interference with other instruments.

* This towel clip was made to our designs by Codman and Shurtleff, Inc., Boston, Mass.

BOOK REVIEWS

ATLAS OF OVARIAN TUMORS. By Gemma Barzilai, M.D., with a preface by Fred W. Stewart, M.D. Grune and Stratton, quarto, 261 pages, illustrated with 58 plates, some in color, $10.00, New York, 1943.

Nothing could furnish better testimony to the enormous interest in the glands of internal secretion in general, and the ovary in particular, than the successive appearance of recent monographs devoted in part or altogether to that organ. In 1940, there was Emil Novak's "Gynecological and Obstetrical Pathology," the best chapters of which concerned tumors of the ovary; in 1942, S. H. Geist's "Ovarian Tumors" appeared, and now, at the beginning of 1943, we have from Dr. Gemma Barzilai an "Atlas of Ovarian Tumors." This handsome monograph has been prepared somewhat in the manner of the magnificent "Atlas du Cancer" brought out in parts during the years 1922 to 1932 by various French pathologists under the auspices of the French Cancer Association and never completed, probably because the depression affected the Rothschild Foundation, which financed it. It consists of a series of reproductions of photomicrographs both in black and white and also in color, with explanatory text conveniently located on the facing page. A useful classification of ovarian neoplasms, with a reasonable histologic and embryologic basis, precedes the text. Most of the names used are conventional and where unusual terms are employed, such as, for example, endosalpingioma, the author gives synonyms in the explanatory text.

The book is addressed particularly to the pathologist who has the difficult assignment of diagnosis by histologic criteria. From this point of view certain weaknesses are apparent. One could not, I believe, from an examination of the illustrations and text, be able to distinguish between theca cell tumor, fibroma and fibrosarcoma. This leads to the general criticism that the detail pictures are not sufficiently magnified to enable one to observe the finer aspects of cellular morphology. Accurate cytologic detail is just as important in the interpretation of ovarian tumors as it is in any other branch of oncology.

The material for the monograph was accumulated in Vienna, Milan, Padua and Istanbul. The text is clearly presented in excellent English, without too many dogmatic statements. It will prove an interesting addition to the library of anyone interested in the subject.

ARTHUR PURDY STOUT, M.D.

OPHTHALMOLOGY AND OTOLARYNGOLOGY—MILITARY SURGICAL MANUALS II. Edited by the National Research Council. W. B. Saunders Company, 331 pp., 188 ills., $4.00. Philadelphia, 1942.

This manual, as stated in the preface, is not intended to be a complete treatise on ophthalmology or otolaryngology. There are only 303 pages, 157 devoted to ophthalmology and 146 to otolaryngology. It was designed to supply certain specialized information concerning injuries and diseases of the eye, ear, nose and throat that are most likely to occur among troops in time of war, either while in training or during actual combat, and especially to aid the physician when so situated that more detailed special knowledge cannot be obtained. The names of the authors of the various chapters of which this book is composed are guarantees for the reliability of the information recorded in its pages and we have no criticism as to the accuracy of the statements made or the recommendation for the treatment and diagnosis suggested. What is said

is reliable, up to date, and authoritative, and there are some chapters that give data frequently omitted from even the most extensive text book. However, we feel that certain aspects of the book are open to criticism.

First, a multitude of authors have apparently interfered with the coordinated arrangement of the subject material, the book being more like the getting together and binding up of a lot of pamphlets than a teaching manual.

Again, there seems to be a difference of purpose between the part on ophthalmology and on otolaryngology as to the clientele for whom the book is intended. From the preface on ophthalmology we quote, "This compendium is not intended to be a treatise on ophthalmology, nor is it designed for the experienced ophthalmologist." One would infer that it was designed for the general practitioner. Whereas in the preface to the part on otolaryngology again we quote, "No attempt has been made to present elementary principles and practice, as it is fair to assume that commissioned otolaryngologists . . . have been trained beyond any such requirement." Perhaps these two statements help explain the differences in the amount and detail of the subject matter in the different chapters and why the ophthalmologic part is so much more detailed and extensive than the otolaryngological. In otolaryngology there are two chapters spent on the diagnosis and treatment of petrositis, a subject which requires the utmost of specialized training, while there is scarely a word concerning the method of examining the ear for functional disturbances. Yet in the part on ophthalmology there are two chapters devoted to functional testing and methods of examining the eye..

Also we wonder why some 42 pages are devoted to facial maxillary surgery which the otolaryngologist in civil practice usually turns over to the maxillofacial surgeon. Useful knowledge yes, but this space could have been more appropriately used for more detailed data concerning real otolaryngologic problems even to the inclusion of some subjects that have been omitted on account of lack of space.

However, in spite of these criticisms, we feel that this manual has a distinct place as a quick reference book for the field surgeon with accurate information which for the greater part is concisely and clearly stated. It is well printed, carefully indexed and the illustrations though not profuse are of distinct help in clarifying operative technic.

GEORGE B. WOOD, M.D.

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